Access DB# 6376/05

SEARCH REQUEST FORM

Scientific and Technical Information Center

Requester's Full Name: P Spivack Examiner #: 70400 Date: 3/24/02
Art Unit: 1614 Phone Number 30 8 4703 Serial Number: 09/889751
Mail Box and Bldg/Room Location: 2D05 Results Format Preferred (circle): PAPER DISK E-MAIL
2D01

If more than one search is submitted, please prioritize searches in order of need:

****

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

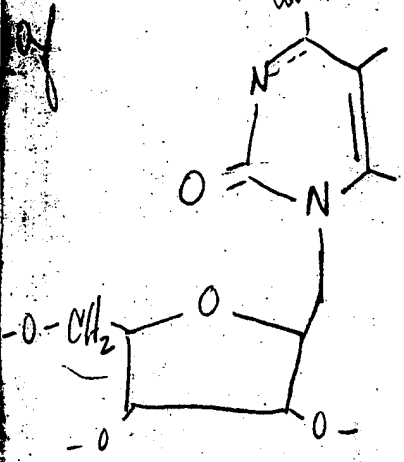
Title of Invention: Treatment of Mitochondrial Disorders

Inventors (please provide full names): Robert K. Naviaux

Earliest Priority Filing Date: 2/23/99

**For Sequence Searches Only* Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.*

Please search:
methods of treating any one of the disorders on the attached sheet comprising administering a compound of



Please eliminate 2002, 2001, 2000 refs.

Point of Contact:
Barb O'Bryen
Technical Information Specialist
STIC CM1 6A05 308-4291

example - trimethyluridine

Thanks

****

STAFF USE ONLY

Searcher: _____
Searcher Phone #: _____
Searcher Location: _____
Date Searcher Picked Up: _____
Date Completed: 3-26-02
Searcher Prep & Review Time: prep 25
Clerical Prep Time: _____
Online Time: 75

Type of Search

NA Sequence (#) _____
AA Sequence (#) _____
Structure (#) 2
Bibliographic _____
Litigation _____
Fulltext _____
Patent Family _____
Other _____

Vendors and cost where applicable

STN 425
Dialog _____
Questel/Orbit _____
Dr.Link _____
Lexis/Nexis _____
Sequence Systems _____
WWW/Internet _____
Other (specify) _____

PTO-1590 (8-01)

$%^STN;HighlightOn=;**HighlightOff=;**
=> fil reg; d ide
FILE 'REGISTRY' ENTERED AT 15:05:53 ON 26 MAR 2002
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STRUCTURE FILE UPDATES: 25 MAR 2002 HIGHEST RN 402820-22-8
DICTIONARY FILE UPDATES: 25 MAR 2002 HIGHEST RN 402820-22-8

TSCA INFORMATION NOW CURRENT THROUGH July 7, 2001

Please note that search-term pricing does apply when conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Calculated physical property data is now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

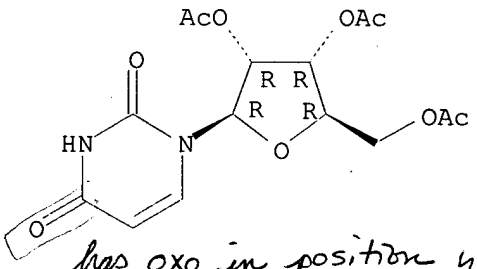
The P indicator for Preparations was not generated for all of the CAS Registry Numbers that were added to the H/Z/CA/CAplus files between 12/27/01 and 1/23/02. Use of the P indicator in online and SDI searches during this period, either directly appended to a CAS Registry Number or by qualifying an L-number with /P, may have yielded incomplete results. As of 1/23/02, the situation has been resolved. Also, note that searches conducted using the PREP role indicator were not affected.

Customers running searches and/or SDIs in the H/Z/CA/CAplus files incorporating CAS Registry Numbers with the P indicator between 12/27/01 and 1/23/02, are encouraged to re-run these strategies. Contact the CAS Help Desk at 1-800-848-6533 in North America or 1-614-447-3698, worldwide, or send an e-mail to help@cas.org for further assistance or to receive a credit for any duplicate searches.

L75 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2002 ACS
RN **4105-38-8** REGISTRY
CN Uridine, 2',3',5'-triacetate (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2',3',5'-Tri-O-acetyluridine
CN 2',3',5'-Triacetyluridine
CN Tri-O-acetyl uridine
FS STEREOSEARCH
DR 293738-13-3
MF C15 H18 N2 O9
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DRUGUPDATES, HODOC*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

This compound was retrieved from inventor's citation

Absolute stereochemistry.



has oxo in position you indicated as "cannot be oxo"

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

161 REFERENCES IN FILE CA (1967 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
161 REFERENCES IN FILE CAPLUS (1967 TO DATE)
4 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> fil capl; d que 178
FILE 'CAPLUS' ENTERED AT 15:30:07 ON 26 MAR 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE COVERS 1907 - 26 Mar 2002 VOL 136 ISS 13
FILE LAST UPDATED: 25 Mar 2002 (20020325/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

CAS roles have been modified effective December 16, 2001. Please check your SDI profiles to see if they need to be revised. For information on CAS roles, enter HELP ROLES at an arrow prompt or use the CAS Roles thesaurus (/RL field) in this file.

The P indicator for Preparations was not generated for all of the CAS Registry Numbers that were added to the CAS files between 12/27/01 and 1/23/02. As of 1/23/02, the situation has been resolved. Searches and/or SDIs in the H/Z/CA/CAplus files incorporating CAS Registry Numbers with the P indicator executed between 12/27/01 and 1/23/02 may be incomplete. See the NEWS message on this topic for more information.

search of triacetyluridine + diseases

L12 577 SEA FILE=HCAPLUS ABB=ON MELAS OR MITOCHONDRIAL(A)ENCEPHALOMYOPATH?
L13 139 SEA FILE=HCAPLUS ABB=ON MERRF OR MYCLONUS(L)EPILEPSY(L)MYOPATH?/OBI
L14 89 SEA FILE=HCAPLUS ABB=ON NARP(W)MILS OR NEUROGENIC (L)ATAXIA(L)PIGMENTOSA/OBI OR LEIGH SYNDROME

L15 134 SEA FILE=HCAPLUS ABB=ON LHON OR LEBERS(L)OPTIC(L)NEUROPATH?/OB I
L16 335 SEA FILE=HCAPLUS ABB=ON MITOCHONDRIAL(A)BLINDNESS OR KSS OR KEARNS(A)SAYRE
L17 272 SEA FILE=HCAPLUS ABB=ON PMPS OR MARROW(A)PANCREAS(A)SYNDROME#
L18 145 SEA FILE=HCAPLUS ABB=ON CPEO OR PROGRESSIVE(L)OPHTHALMOPLEG?/O BI
L19 56 SEA FILE=HCAPLUS ABB=ON (ALPER? OR MTDNA OR MT DNA)(L)SYNDROME #/OBI
L20 433 SEA FILE=HCAPLUS ABB=ON (CYTOCHROME(1W)OXIDASE OR COX OR ADENINE NUCLEOTIDE TRANSLOCATOR OR ANT OR PYRUVATE DEHYDROGENAS E OR PDH)(2A)DEFICIENC?
L21 147 SEA FILE=HCAPLUS ABB=ON LACTIC(A)ACIDEM?
L22 13 SEA FILE=HCAPLUS ABB=ON (ETHYLMALONIC OR METHYL GLUTACONIC)(W) ACIDUR?
L23 863 SEA FILE=HCAPLUS ABB=ON REFRACTORY(L)EPILEPSY/OBI OR ASPERGER? SYNDROME OR AUTISM OR CEREBRAL PALSY OR DYSLEX?
L24 878 SEA FILE=HCAPLUS ABB=ON ADHD OR ATTENTION DEFICIT
L25 171 SEA FILE=HCAPLUS ABB=ON (COMPLEX(W)(I OR II OR SDH OR III OR IV OR V))(2A)DEFICIENC?
L26 5860 SEA FILE=HCAPLUS ABB=ON THROMBOCYTOPENI? OR LEUKEMIA SYNDROME
L27 12 SEA FILE=HCAPLUS ABB=ON MNGIE OR MITROCHONDRIAL MYOPATH?(L)NEU ROPATH?(L)EPILEPSY/OBI
L28 1 SEA FILE=HCAPLUS ABB=ON MARIAHS OR MITROCHONDRIAL(L)ATAXIA(L)I NFECTION#(L)APHASI?/OBI
L29 7 SEA FILE=HCAPLUS ABB=ON (ND6 OR ND 6)(L)DYSTONI?/OBI
L30 32 SEA FILE=HCAPLUS ABB=ON CYCLIC(A) VOMITING
L31 86114 SEA FILE=HCAPLUS ABB=ON ?DIABET?
L32 25 SEA FILE=HCAPLUS ABB=ON URIDINE RESPONSIVE NEUROLOGIC SYNDROME OR URNS
L33 50 SEA FILE=HCAPLUS ABB=ON STRIATAL NECROSIS OR FBSN OR AMINOGLYC OSIDE#(L)DEAFNESS/OBI
L34 1331 SEA FILE=HCAPLUS ABB=ON DILATED(A)CARDIOMYOPATH?
L35 47 SEA FILE=HCAPLUS ABB=ON SPLENIC(A)LYMPHOMA#
L36 45 SEA FILE=HCAPLUS ABB=ON WOLFRAM? SYNDROME
L37 56 SEA FILE=HCAPLUS ABB=ON MITOCHONDRIAL(L)(DNA OR DEOXYRIBONUCLE IC)(L)DELETION(L)SYNDROME#/OBI
L38 6817 SEA FILE=HCAPLUS ABB=ON (HEAD OR BRAIN)(L)(TRAUMA? OR INJUR?)/OBI
L39 1852 SEA FILE=HCAPLUS ABB=ON CEREBRAL(L)EDEM?
L40 15736 SEA FILE=HCAPLUS ABB=ON STROKE
L41 7761 SEA FILE=HCAPLUS ABB=ON REPERFUSION(A)INJUR?
L42 21659 SEA FILE=HCAPLUS ABB=ON ALZHEIMER?
L43 14096 SEA FILE=HCAPLUS ABB=ON ?PARKINSON?
L44 131 SEA FILE=HCAPLUS ABB=ON HEPATORENAL SYNDROME
L45 84 SEA FILE=HCAPLUS ABB=ON LEWY BODY(A)DEMENT?
L46 6043 SEA FILE=HCAPLUS ABB=ON HUNTINGTON? OR AMYOTROPHIC LATERAL OR GEHRIG? OR ACUTE(A)LIVER FAILURE
L47 2256 SEA FILE=HCAPLUS ABB=ON NASH OR (NONALCOHOLIC OR NON ALCOHOLIC)(A)STEATOHEPATITIS
L48 1503 SEA FILE=HCAPLUS ABB=ON ANTIMETAS? OR ANTI METAS? OR PRODIFFER ENTIAT?(L)(CANCER? OR TUMOR OR NEOPLAS?)/OBI
L49 4720 SEA FILE=HCAPLUS ABB=ON CONGESTIVE(A)(HEART OR CARDIAC)(W)FAIL URE
L50 5 SEA FILE=HCAPLUS ABB=ON ATRIAL(A)FIBRILATION#
L51 38 SEA FILE=HCAPLUS ABB=ON WOLFF(L)WHITE(L)SYNDROME
L52 3191 SEA FILE=HCAPLUS ABB=ON MIGRAINE#
L53 791 SEA FILE=HCAPLUS ABB=ON IRRITABLE BOWEL
L54 119 SEA FILE=HCAPLUS ABB=ON MYOCARDIAL INFARCT?(3A)NON Q WAVE#
L55 1100 SEA FILE=HCAPLUS ABB=ON (PREMENSTRUAL OR HEPATORENAL OR ANTIPHOSPHOLIPID OR ANTI PHOSPHOLIPID OR PHOSPHOLIPID(2A)ANTIBO

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               D?)(L) SYNDROME/OBI
L56         2967 SEA FILE=HCAPLUS ABB=ON  ECLAMP? OR PREECLAMP?
L57            1 SEA FILE=HCAPLUS ABB=ON  OOPAUS?
L58        15787 SEA FILE=HCAPLUS ABB=ON  (ISCHEMI? OR ISCHAEMI?)(L)(HEART OR
               CARDIAC)/OBI
L59         5794 SEA FILE=HCAPLUS ABB=ON  ANGINA OR ANTIANGINA
L60           58 SEA FILE=HCAPLUS ABB=ON  SHY(A) DRAGER
L61          116 SEA FILE=HCAPLUS ABB=ON  DYSAUTONOMI?
L62          829 SEA FILE=HCAPLUS ABB=ON  RENAL TUBULAR(A) ACIDOSIS OR HEART
               BLOCK?
L63           13 SEA FILE=HCAPLUS ABB=ON  ?ISOBUTYRIC?(3A)ACIDURI?
L72           32 SEA FILE=HCAPLUS ABB=ON  TRIACETYLURIDINE
L75            1 SEA FILE=REGISTRY ABB=ON  4105-38-8
L76          177 SEA FILE=HCAPLUS ABB=ON  L75 OR L72 OR TRIACETATE URIDINE OR
               (TRI O)(W)(ACETYLURIDINE OR ACETYL URIDINE)
L77          157 SEA FILE=HCAPLUS ABB=ON  L76 NOT PY>1999
L78            0 SEA FILE=HCAPLUS ABB=ON  L77 AND (L12 OR L13 OR L14 OR L15 OR
               L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR
               L25 OR L26 OR L27 OR L28 OR L29 OR L30 OR L31 OR L32 OR L33 OR
               L34 OR L35 OR L36 OR L37 OR L38 OR L39 OR L40 OR L41 OR L42 OR
               L43 OR L44 OR L45 OR L46 OR L47 OR L48 OR L49 OR L50 OR L51 OR
               L52 OR L53 OR L54 OR L55 OR L56 OR L57 OR L58 OR L59 OR L60 OR
               L61 OR L62 OR L63)


=> fil cancer medl caba drugu biosis biotechno embase uspatf
FILE 'CANCERLIT' ENTERED AT 15:30:20 ON 26 MAR 2002

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=> d que l97; d que l104
L72           32 SEA FILE=HCAPLUS ABB=ON  TRIACETYLURIDINE
L75            1 SEA FILE=REGISTRY ABB=ON  4105-38-8
L92          108 SEA L75 OR L72 OR TRIACETATE URIDINE OR (TRI O)(W)(ACETYLURIDIN
               E OR ACETYL URIDINE)
L93           57 SEA TRI(1W)(((ACETYL OR ACETATE) (W) URIDINE) OR ACETYLURIDINE)

L94          108 SEA L92 OR L93
L95           62 SEA L94 NOT PY>1999
L96       400242 SEA MITOCHONDRI?
L97            1 SEA L95 AND L96



L12          577 SEA FILE=HCAPLUS ABB=ON  MELAS OR MITOCHONDRIAL(A)ENCEPHALOMYOP
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		ATH?
L13	139	SEA FILE=HCAPLUS ABB=ON MERRF OR MYCLONUS(L)EPILEPSY(L)MYOPATH ?/OBI
L14	89	SEA FILE=HCAPLUS ABB=ON NARP(W)MILS OR NEUROGENIC (L)ATAXIA(L) PIGMENTOSA/OBI OR LEIGH SYNDROME
L15	134	SEA FILE=HCAPLUS ABB=ON LHON OR LEBERS(L)OPTIC(L)NEUROPATH?/OB I
L16	335	SEA FILE=HCAPLUS ABB=ON MITOCHONDRIAL(A)BLINDNESS OR KSS OR KEARNS(A)SAYRE
L17	272	SEA FILE=HCAPLUS ABB=ON PMPS OR MARROW(A)PANCREAS(A)SYNDROME#
L18	145	SEA FILE=HCAPLUS ABB=ON CPEO OR PROGRESSIVE(L)OPHTHALMOPLEG?/O BI
L19	56	SEA FILE=HCAPLUS ABB=ON (ALPER? OR MTDNA OR MT DNA)(L)SYNDROME #/OBI
L20	433	SEA FILE=HCAPLUS ABB=ON (CYTOCHROME(1W)OXIDASE OR COX OR ADENINE NUCLEOTIDE TRANSLOCATOR OR ANT OR PYRUVATE DEHYDROGENAS E OR PDH)(2A)DEFICIENC?
L21	147	SEA FILE=HCAPLUS ABB=ON LACTIC(A)ACIDEM?
L22	13	SEA FILE=HCAPLUS ABB=ON (ETHYLMALONIC OR METHYL GLUTACONIC)(W) ACIDUR?
L23	863	SEA FILE=HCAPLUS ABB=ON REFRACTORY(L)EPILEPSY/OBI OR ASPERGER? SYNDROME OR AUTISM OR CEREBRAL PALSY OR DYSLEX?
L24	878	SEA FILE=HCAPLUS ABB=ON ADHD OR ATTENTION DEFICIT
L25	171	SEA FILE=HCAPLUS ABB=ON (COMPLEX(W)(I OR II OR SDH OR III OR IV OR V))(2A)DEFICIENC?
L26	5860	SEA FILE=HCAPLUS ABB=ON THROMBOCYTOPENI? OR LEUKEMIA SYNDROME
L27	12	SEA FILE=HCAPLUS ABB=ON MNGIE OR MITROCHONDRIAL MYOPATH?(L)NEU ROPATH?(L)EPILEPSY/OBI
L28	1	SEA FILE=HCAPLUS ABB=ON MARIAHS OR MITROCHONDRIAL(L)ATAXIA(L)I NFECTION#(L)APHASI?/OBI
L29	7	SEA FILE=HCAPLUS ABB=ON (ND6 OR ND 6)(L)DYSTONI?/OBI
L30	32	SEA FILE=HCAPLUS ABB=ON CYCLIC(A) VOMITING
L31	86114	SEA FILE=HCAPLUS ABB=ON ?DIABET?
L32	25	SEA FILE=HCAPLUS ABB=ON URIDINE RESPONSIVE NEUROLOGIC SYNDROME OR URNS
L33	50	SEA FILE=HCAPLUS ABB=ON STRIATAL NECROSIS OR FBSN OR AMINOGLYC OSIDE#(L)DEAFNESS/OBI
L34	1331	SEA FILE=HCAPLUS ABB=ON DILATED(A)CARDIOMYOPATH?
L35	47	SEA FILE=HCAPLUS ABB=ON SPLENIC(A)LYMPHOMA#
L36	45	SEA FILE=HCAPLUS ABB=ON WOLFRAM? SYNDROME
L37	56	SEA FILE=HCAPLUS ABB=ON MITOCHONDRIAL(L)(DNA OR DEOXYRIBONUCLE IC)(L)DELETION(L)SYNDROME#/OBI
L38	6817	SEA FILE=HCAPLUS ABB=ON (HEAD OR BRAIN)(L)(TRAUMA? OR INJUR?)/OBI
L39	1852	SEA FILE=HCAPLUS ABB=ON CEREBRAL(L)EDEM?
L40	15736	SEA FILE=HCAPLUS ABB=ON STROKE
L41	7761	SEA FILE=HCAPLUS ABB=ON REPERFUSION(A)INJUR?
L42	21659	SEA FILE=HCAPLUS ABB=ON ALZHEIMER?
L43	14096	SEA FILE=HCAPLUS ABB=ON ?PARKINSON?
L44	131	SEA FILE=HCAPLUS ABB=ON HEPATORENAL SYNDROME
L45	84	SEA FILE=HCAPLUS ABB=ON LEWY BODY(A)DEMENT?
L46	6043	SEA FILE=HCAPLUS ABB=ON HUNTINGTON? OR AMYOTROPHIC LATERAL OR GEHRIG? OR ACUTE(A)LIVER FAILURE
L47	2256	SEA FILE=HCAPLUS ABB=ON NASH OR (NONALCOHOLIC OR NON ALCOHOLIC)(A)STEATOHEPATITIS
L48	1503	SEA FILE=HCAPLUS ABB=ON ANTIMETAS? OR ANTI METAS? OR PRODIFFER ENTIAT?(L)(CANCER? OR TUMOR OR NEOPLAS?)/OBI
L49	4720	SEA FILE=HCAPLUS ABB=ON CONGESTIVE(A)(HEART OR CARDIAC)(W)FAIL URE
L50	5	SEA FILE=HCAPLUS ABB=ON ATRIAL(A)FIBRILATION#
L51	38	SEA FILE=HCAPLUS ABB=ON WOLFF(L)WHITE(L)SYNDROME

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L52         3191 SEA FILE=HCAPLUS ABB=ON  MIGRAINE#
L53          791 SEA FILE=HCAPLUS ABB=ON  IRRITABLE BOWEL
L54          119 SEA FILE=HCAPLUS ABB=ON  MYOCARDIAL INFARCT?(3A)NON Q WAVE#
L55         1100 SEA FILE=HCAPLUS ABB=ON  (PREMENSTRUAL OR HEPATORENAL OR
                 ANTIPHOSPHOLIPID OR ANTI PHOSPHOLIPID OR PHOSPHOLIPID(2A)ANTIBO
                 D?)(L) SYNDROME/OBI
L56         2967 SEA FILE=HCAPLUS ABB=ON  ECLAMP? OR PREECLAMP?
L57            1 SEA FILE=HCAPLUS ABB=ON  OOPAUS?
L58        15787 SEA FILE=HCAPLUS ABB=ON  (ISCHEMI? OR ISCHAEMI?)(L)(HEART OR
                 CARDIAC)/OBI
L59         5794 SEA FILE=HCAPLUS ABB=ON  ANGINA OR ANTIANGINA
L60           58 SEA FILE=HCAPLUS ABB=ON  SHY(A) DRAGER
L61          116 SEA FILE=HCAPLUS ABB=ON  DYSAUTONOMI?
L62          829 SEA FILE=HCAPLUS ABB=ON  RENAL TUBULAR(A) ACIDOSIS OR HEART
                 BLOCK?
L63           13 SEA FILE=HCAPLUS ABB=ON  ?ISOBUTYRIC?(3A)ACIDURI?
L72           32 SEA FILE=HCAPLUS ABB=ON  TRIACETYLURIDINE
L75            1 SEA FILE=REGISTRY ABB=ON  4105-38-8
L92          108 SEA L75 OR L72 OR TRIACETATE URIDINE OR (TRI O)(W)(ACETYLURIDIN
                 E OR ACETYL URIDINE)
L93           57 SEA TRI(1W)(((ACETYL OR ACETATE) (W) URIDINE) OR ACETYLURIDINE)

L94          108 SEA L92 OR L93
L95           62 SEA L94 NOT PY>1999
L98        81799 SEA (L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR
                 L20 OR L21 OR L22 OR L23 OR L24 OR L25)
L99       779922 SEA (L26 OR L27 OR L28 OR L29 OR L30 OR L*** OR L31 OR L32 OR
                 L33 OR L34 OR L35 OR L36)
L104            6 SEA L95 AND (L98 OR L99 OR L100 OR L101 OR L102 OR L103)


=> s l97 or l104
L105            6 L97 OR L104

=> dup rem l105
PROCESSING COMPLETED FOR L105
L106            6 DUP REM L105 (0 DUPLICATES REMOVED)
                  ANSWER '1' FROM FILE DRUGU
                  ANSWERS '2-6' FROM FILE USPATFULL

=> d ibib ab hitrn l106 1-6

L106  ANSWER 1 OF 6  DRUGU  COPYRIGHT 2002 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1994-49868  DRUGU   T S
TITLE:            Oral triacetyluridine (TAU) as a rescue agent for
                  5-fluorouracil (5FU): phase I and pharmacological study.
AUTHOR:           Schwartz G; Kelsen D; Saltz L; Kemeny N; Caspar E; Toomasi F
CORPORATE SOURCE: Memorial-Sloan-Kettering-Cancer-Cent.; Pro-Neuron
LOCATION:         New York, New York, Rockville, Maryland, United States
SOURCE:           Proc.Am.Soc.Clin.Oncol. (13, 30 Meet., 134, 1994)       ISSN:
AVAIL. OF DOC.:   Memorial Sloan-Kettering Cancer Center, New York, NY 10021,
                  U.S.A. (10 authors).
LANGUAGE:         English
DOCUMENT TYPE:    Journal
FIELD AVAIL.:     AB; LA; CT; MPC
FILE SEGMENT:     Literature
AB    Uridine levels of 50-100 uM can ameliorate fluorouracil (5FU) induced
      hematopoietic and GI toxicity in animals. Triacetyluridine
      (TAU), an p.o. prodrug of uridine, preclinically acheives 8-fold higher
      uridine levels than equimolar doses of uridine. The Cmax, Cmin and AUC
      of uridine were calculated after the administration of bolus 5FU + p.o.
      TAU in 29 patients with incurable cancers. With escalating doses of
      bolus 5FU + TAU (3.3 g), neutropenia was seen. With escalating doses of
```

5FU and TAU (6.6 g; ensuring Cmax and Cmin uridine levels over 50 uM), there was no hematologic (measured by WBC, absolute neutrophil and platelet count) or GI toxicity. Sustained uridine levels over 50 uM, which are achieved with 6.6 g of TAU, prevent the toxicity of bolus 5FU seen on this wkly schedule. (conference abstract).

L106 ANSWER 2 OF 6 USPATFULL
ACCESSION NUMBER: 1998:98932 USPATFULL
TITLE: DHA-pharmaceutical agent conjugates of taxanes
INVENTOR(S): Shashoua, Victor E., Brookline, MA, United States
Swindell, Charles S., Merion, PA, United States
Webb, Nigel L., Bryn Mawr, PA, United States
Bradley, Matthews O., Laytonsville, MD, United States
PATENT ASSIGNEE(S): Neuromedica, Inc., Conshohocken, PA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5795909		19980818	
APPLICATION INFO.:	US 1996-651312		19960522	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jarvis, William R. A.
LEGAL REPRESENTATIVE: Wolf, Greenfield & Sacks, P.C.
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Figure(s); 14 Drawing Page(s)
LINE COUNT: 2451
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides conjugates of cis-docosahexaenoic acid and taxanes useful in treating cell proliferative disorders. Conjugates of paclitaxel and docetaxel are preferred.

L106 ANSWER 3 OF 6 USPATFULL
ACCESSION NUMBER: 1998:72607 USPATFULL
TITLE: Pharmaceutical compositions containing deoxyribonucleosides for wound healing
INVENTOR(S): von Borstel, Reid Warren, Kensington, MD, United States
Bamat, Michael Kevin, Chevy Chase, MD, United States
PATENT ASSIGNEE(S): Pro-Neuron, Inc., Gaithersburg, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5770582		19980623	
APPLICATION INFO.:	US 1995-419767		19950410	(8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-96407, filed on 26 Jul 1993, now abandoned which is a division of Ser. No. US 1992-911379, filed on 13 Jul 1992, now patented, Pat. No. US 5246708 which is a continuation of Ser. No. US 1989-341925, filed on 21 Apr 1989, now abandoned which is a continuation-in-part of Ser. No. US 1988-186031, filed on 25 Apr 1988, now abandoned which is a continuation-in-part of Ser. No. US 1987-115923, filed on 28 Oct 1987, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Kunz, Gary L.
LEGAL REPRESENTATIVE: Nixon & Vanderhye
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 1132

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to compositions comprising 2'-deoxyribonucleosides. The invention also relates to methods of accelerating the healing of wounds, abrasions, cuts, incisions, and superficial burns induced by heat, sunlight, chemical agents, or infections, and methods for ameliorating the effects of aging of the epidermal tissues comprising administering the compositions of the present invention to an animal.
IT **4105-38-8P**, 2',3',5'-Tri-O-acetyluridine
(prepn. of, as drug)

L106 ANSWER 4 OF 6 USPATFULL
ACCESSION NUMBER: 96:113912 USPATFULL
TITLE: Acylated uridine and cytidine for elevating tissue uridine and cytidine
INVENTOR(S): von Borstel, Reid, Kensington, MD, United States
Bamat, Michael K., Chevy Chase, MD, United States
PATENT ASSIGNEE(S): Pro-Neuron, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5583117		19961210
APPLICATION INFO.:	US 1993-140475		19931025 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1991-737913, filed on 29 Jul 1991, now abandoned which is a continuation of Ser. No. US 1987-115929, filed on 28 Oct 1987, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Kunz, Gary L.
LEGAL REPRESENTATIVE: Nixon & Vanderhye P.C.
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 1658
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB General methods for delivering exogenous cytidine or uridine to the tissue of an animal comprising the administration of acylated cytidine or acylated uridine, respectively, are disclosed. Methods of treating myocardial infarction and cardiac insufficiency comprising the administration of acylated cytidine or acylated uridine, are also disclosed.
IT **4105-38-8P**, 2',3',5'-Tri-O-acetyluridine
(prepn. of, as drug)

L106 ANSWER 5 OF 6 USPATFULL
ACCESSION NUMBER: 95:105828 USPATFULL
TITLE: Method of delivering exogenous uridine or cytidine using acylated uridine or cytidine
INVENTOR(S): von Borstel, Reld W., Darnestown, MD, United States
Bamat, Michael K., Darnestown, MD, United States
PATENT ASSIGNEE(S): Pro-Neuron, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5470838		19951128
APPLICATION INFO.:	US 1992-997657		19921230 (7)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1990-438493, filed on 26 Jun 1990, now abandoned which is a continuation-in-part of Ser. No. US 1987-115929, filed on 28 Oct 1987, now abandoned
DOCUMENT TYPE: Utility

FILE SEGMENT: Granted
PRIMARY EXAMINER: Robinson, Douglas W.
ASSISTANT EXAMINER: Kunz, Gary L.
LEGAL REPRESENTATIVE: Nixon & Vanderhye
NUMBER OF CLAIMS: 61
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 1745
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods of delivering exogenous uridine or cytidine to the tissue of an animal comprising the administration of acyl derivatives of uridine or cytidine, respectively, are disclosed. Also disclosed are methods for treating cardiac insufficiency, myocardial infarction, cirrhosis of the liver, each comprising administration of acyl derivatives of uridine or cytidine.

IT **4105-38-8P**, 2',3',5'-Tri-O-acetyluridine
(prepn. of, as drug)

L106 ANSWER 6 OF 6 USPATFULL
ACCESSION NUMBER: 93:78557 USPATFULL
TITLE: Methods for promoting wound healing with deoxyribonucleosides
INVENTOR(S): von Borstel, Reid W., Kensington, MD, United States
Bamat, Michael K., Chevy Chase, MD, United States
PATENT ASSIGNEE(S): Pro-Neuron, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5246708		19930921
APPLICATION INFO.:	US 1992-911379		19920713 (7)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1989-341925, filed on 21 Apr 1989, now abandoned which is a continuation-in-part of Ser. No. US 1988-186031, filed on 25 Apr 1988, now abandoned which is a continuation-in-part of Ser. No. US 1987-115923, filed on 28 Oct 1987, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Rollins, John W.
ASSISTANT EXAMINER: Kunz, Gary L.
LEGAL REPRESENTATIVE: Nixon & Vanderhye
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 25
NUMBER OF DRAWINGS: 9 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 1043
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for promoting wound healing in animals by administering compositions containing two or more of the following 2'-deoxyribonucleosides: 2'-deoxyadenosine, 2'-deoxyguanosine, 2'-deoxy-cytidine, or thymidine. 3'- and 5'-phosphate derivatives of these 2'-deoxynucleosides are also effective in promoting wound healing.

IT **4105-38-8P**, 2',3',5'-Tri-O-acetyluridine
(prepn. of, as drug)

```
=> fil reg; d stat que l9
FILE 'REGISTRY' ENTERED AT 15:31:12 ON 26 MAR 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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STRUCTURE FILE UPDATES:   25 MAR 2002  HIGHEST RN 402820-22-8
DICTIONARY FILE UPDATES:  25 MAR 2002  HIGHEST RN 402820-22-8

TSCA INFORMATION NOW CURRENT THROUGH July 7, 2001

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Crossover limits have been increased.  See HELP CROSSOVER for details.

Calculated physical property data is now available.  See HELP PROPERTIES
for more information.  See STNote 27, Searching Properties in the CAS
Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

The P indicator for Preparations was not generated for all of the
CAS Registry Numbers that were added to the H/Z/CA/CAplus files between
12/27/01 and 1/23/02.  Use of the P indicator in online and SDI searches
during this period, either directly appended to a CAS Registry Number
or by qualifying an L-number with /P, may have yielded incomplete results.
As of 1/23/02, the situation has been resolved.  Also, note that searches
conducted using the PREP role indicator were not affected.

Customers running searches and/or SDIs in the H/Z/CA/CAplus files
incorporating CAS Registry Numbers with the P indicator between 12/27/01
and 1/23/02, are encouraged to re-run these strategies.  Contact the
CAS Help Desk at 1-800-848-6533 in North America or 1-614-447-3698,
worldwide, or send an e-mail to help@cas.org for further assistance or to
receive a credit for any duplicate searches.

L1                STR
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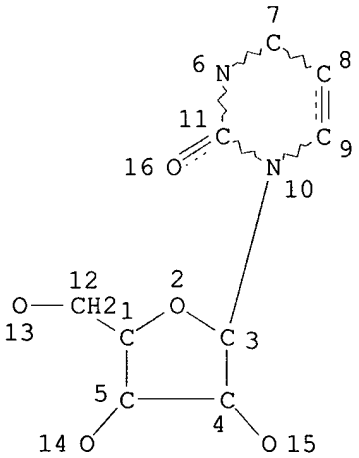

full file search done on this structure

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NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
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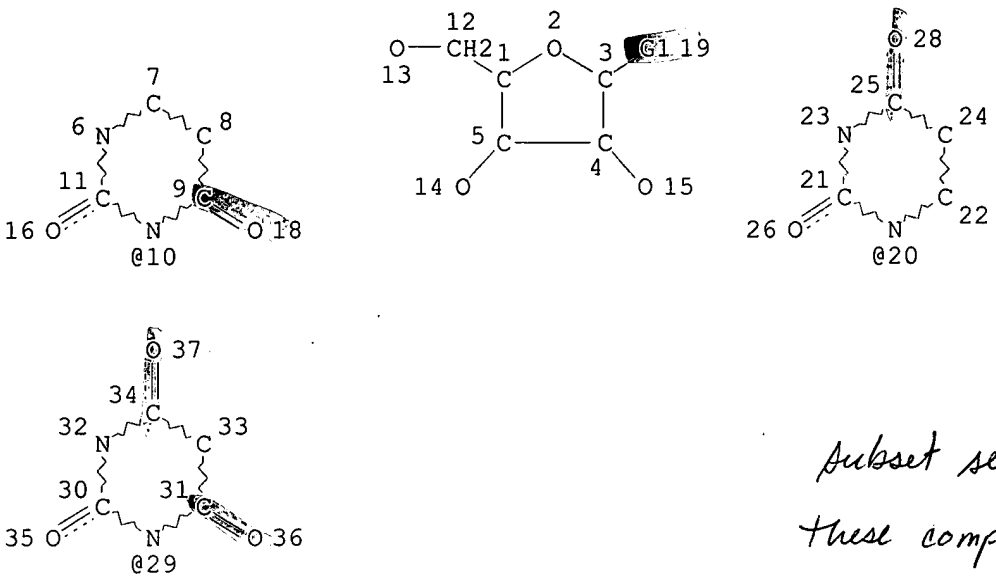
NUMBER OF NODES IS 16

STEREO ATTRIBUTES: NONE
L3 26337 SEA FILE=REGISTRY SSS FUL L1
L7 STR



Subset search done, removing these compounds from the answer set (oxo at "forbidden" positions)

VAR G1=10/20/29
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 35

STEREO ATTRIBUTES: NONE
L9 10019 SEA FILE=REGISTRY SUB=L3 SSS FUL (L1 NOT L7)

100.0% PROCESSED 26337 ITERATIONS 10019 ANSWERS
SEARCH TIME: 00.00.02

=> fil hcapl
FILE 'HCAPLUS' ENTERED AT 15:32:44 ON 26 MAR 2002

FILE COVERS 1907 - 26 Mar 2002 VOL 136 ISS 13
FILE LAST UPDATED: 25 Mar 2002 (20020325/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

CAS roles have been modified effective December 16, 2001. Please check your SDI profiles to see if they need to be revised. For information on CAS roles, enter HELP ROLES at an arrow prompt or use the CAS Roles thesaurus (/RL field) in this file.

The P indicator for Preparations was not generated for all of the CAS Registry Numbers that were added to the CAS files between 12/27/01 and 1/23/02. As of 1/23/02, the situation has been resolved. Searches and/or SDIs in the H/Z/CA/CAplus files incorporating CAS Registry Numbers with the P indicator executed between 12/27/01 and 1/23/02 may be incomplete. See the NEWS message on this topic for more information.

compounds from structure search + diseases

```
=> d que l67 nos ; d que nos 185; s l67 or 185
L1              STR
L3          26337 SEA FILE=REGISTRY SSS FUL L1
L7              STR
L9          10019 SEA FILE=REGISTRY SUB=L3 SSS FUL (L1 NOT L7)
L12           577 SEA FILE=HCAPLUS ABB=ON  MELAS OR MITOCHONDRIAL(A)ENCEPHALOMYOP
                  ATH?
L13           139 SEA FILE=HCAPLUS ABB=ON  MERRF OR MYCLONUS(L)EPILEPSY(L)MYOPATH
                  ?/OBI
L14            89 SEA FILE=HCAPLUS ABB=ON  NARP(W)MILS OR NEUROGENIC (L)ATAXIA(L)
                  PIGMENTOSA/OBI OR LEIGH SYNDROME
L15           134 SEA FILE=HCAPLUS ABB=ON  LHON OR LEBERS(L)OPTIC(L)NEUROPATH?/OB
                  I
L16           335 SEA FILE=HCAPLUS ABB=ON  MITOCHONDRIAL(A)BLINDNESS OR KSS OR
                  KEARNS(A)SAYRE
L17           272 SEA FILE=HCAPLUS ABB=ON  PMPS OR MARROW(A)PANCREAS(A)SYNDROME#

L18           145 SEA FILE=HCAPLUS ABB=ON  CPEO OR PROGRESSIVE(L)OPHTHALMOPLEG?/O
                  BI
L19            56 SEA FILE=HCAPLUS ABB=ON  (ALPER? OR MTDNA OR MT DNA)(L)SYNDROME
                  #/OBI
L20           433 SEA FILE=HCAPLUS ABB=ON  (CYTOCHROME(1W)OXIDASE OR COX OR
                  ADENINE NUCLEOTIDE TRANSLOCATOR OR ANT OR PYRUVATE DEHYDROGENAS
                  E OR PDH)(2A)DEFICIENC?
L21           147 SEA FILE=HCAPLUS ABB=ON  LACTIC(A)ACIDEM?
L22            13 SEA FILE=HCAPLUS ABB=ON  (ETHYLMALONIC OR METHYL GLUTACONIC)(W)
                  ACIDUR?
L23           863 SEA FILE=HCAPLUS ABB=ON  REFRACTORY(L)EPILEPSY/OBI OR ASPERGER?
                   SYNDROME OR AUTISM OR CEREBRAL PALSY OR DYSLEX?
L24           878 SEA FILE=HCAPLUS ABB=ON  ADHD OR ATTENTION DEFICIT
L25           171 SEA FILE=HCAPLUS ABB=ON  (COMPLEX(W)(I OR II OR SDH OR III OR
                  IV OR V))(2A)DEFICIENC?
L27            12 SEA FILE=HCAPLUS ABB=ON  MNGIE OR MITROCHONDRIAL MYOPATH?(L)NEU
                  ROPATH?(L)EPILEPSY/OBI
L28             1 SEA FILE=HCAPLUS ABB=ON  MARIAHS OR MITROCHONDRIAL(L)ATAXIA(L)I
                  NFECTION#(L)APHASI?/OBI
L29             7 SEA FILE=HCAPLUS ABB=ON  (ND6 OR ND 6)(L)DYSTONI?/OBI
L30            32 SEA FILE=HCAPLUS ABB=ON  CYCLIC(A) VOMITING
L32            25 SEA FILE=HCAPLUS ABB=ON  URIDINE RESPONSIVE NEUROLOGIC
                  SYNDROME OR URNS
L33            50 SEA FILE=HCAPLUS ABB=ON  STRIATAL NECROSIS OR FBSN OR AMINOGLYC
                  OSIDE#(L)DEAFNESS/OBI
L34          1331 SEA FILE=HCAPLUS ABB=ON  DILATED(A)CARDIOMYOPATH?
L35            47 SEA FILE=HCAPLUS ABB=ON  SPLENIC(A)LYMPHOMA#
L36            45 SEA FILE=HCAPLUS ABB=ON  WOLFRAM? SYNDROME
L37            56 SEA FILE=HCAPLUS ABB=ON  MITOCHONDRIAL(L)(DNA OR DEOXYRIBONUCLE
                  IC)(L)DELETION(L)SYNDROME#/OBI
L39          1852 SEA FILE=HCAPLUS ABB=ON  CEREBRAL(L)EDEM?
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L41	7761	SEA FILE=HCAPLUS ABB=ON REPERFUSION(A)INJUR?
L44	131	SEA FILE=HCAPLUS ABB=ON HEPATORENAL SYNDROME
L45	84	SEA FILE=HCAPLUS ABB=ON LEWY BODY(A)DEMENT?
L47	2256	SEA FILE=HCAPLUS ABB=ON NASH OR (NONALCOHOLIC OR NON ALCOHOLIC)(A)STEATOHEPATITIS
L49	4720	SEA FILE=HCAPLUS ABB=ON CONGESTIVE(A)(HEART OR CARDIAC)(W)FAILURE
L50	5	SEA FILE=HCAPLUS ABB=ON ATRIAL(A)FIBRILATION#
L51	38	SEA FILE=HCAPLUS ABB=ON WOLFF(L)WHITE(L)SYNDROME
L52	3191	SEA FILE=HCAPLUS ABB=ON MIGRAINE#
L53	791	SEA FILE=HCAPLUS ABB=ON IRRITABLE BOWEL
L54	119	SEA FILE=HCAPLUS ABB=ON MYOCARDIAL INFARCT?(3A)NON Q WAVE#
L55	1100	SEA FILE=HCAPLUS ABB=ON (PREMENSTRUAL OR HEPATORENAL OR ANTIPHOSPHOLIPID OR ANTI PHOSPHOLIPID OR PHOSPHOLIPID(2A)ANTIBOD?)(L) SYNDROME/OBI
L56	2967	SEA FILE=HCAPLUS ABB=ON ECLAMP? OR PREECLAMP?
L57	1	SEA FILE=HCAPLUS ABB=ON OOPAUS?
L59	5794	SEA FILE=HCAPLUS ABB=ON ANGINA OR ANTIANGINA
L60	58	SEA FILE=HCAPLUS ABB=ON SHY(A) DRAGER
L61	116	SEA FILE=HCAPLUS ABB=ON DYSAUTONOMI?
L62	829	SEA FILE=HCAPLUS ABB=ON RENAL TUBULAR(A) ACIDOSIS OR HEART BLOCK?
L63	13	SEA FILE=HCAPLUS ABB=ON ?ISOBUTYRIC?(3A)ACIDURI?
L64	21673	SEA FILE=HCAPLUS ABB=ON L9
L65	19704	SEA FILE=HCAPLUS ABB=ON L64 NOT PY>1999
L67	27	SEA FILE=HCAPLUS ABB=ON L65 AND ((L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25) OR (L27 OR L28 OR L29 OR L30) OR (L32 OR L33 OR L34 OR L35 OR L36 OR L37) OR L39 OR L41 OR L44 OR L45 OR L47 OR (L49 OR L50 OR L51 OR L52 OR L53 OR L54 OR L55 OR L56 OR L57) OR (L59 OR L60 OR L61 OR L62 OR L63))

L1		STR
L3	26337	SEA FILE=REGISTRY SSS FUL L1
L7		STR
L9	10019	SEA FILE=REGISTRY SUB=L3 SSS FUL (L1 NOT L7)
L26	5860	SEA FILE=HCAPLUS ABB=ON THROMBOCYTOPENI? OR LEUKEMIA SYNDROME
L31	86114	SEA FILE=HCAPLUS ABB=ON ?DIABET?
L38	6817	SEA FILE=HCAPLUS ABB=ON (HEAD OR BRAIN)(L)(TRAUMA? OR INJUR?)/OBI
L40	15736	SEA FILE=HCAPLUS ABB=ON STROKE
L42	21659	SEA FILE=HCAPLUS ABB=ON ALZHEIMER?
L43	14096	SEA FILE=HCAPLUS ABB=ON ?PARKINSON?
L46	6043	SEA FILE=HCAPLUS ABB=ON HUNTINGTON? OR AMYOTROPHIC LATERAL OR GEHRIG? OR ACUTE(A)LIVER FAILURE
L48	1503	SEA FILE=HCAPLUS ABB=ON ANTIMETAS? OR ANTI METAS? OR PRODIFFERENTIAT?(L)(CANCER? OR TUMOR OR NEOPLAS?)/OBI
L58	15787	SEA FILE=HCAPLUS ABB=ON (ISCHEMI? OR ISCHAEMI?)(L)(HEART OR CARDIAC)/OBI
L64	21673	SEA FILE=HCAPLUS ABB=ON L9
L65	19704	SEA FILE=HCAPLUS ABB=ON L64 NOT PY>1999
L83	126246	SEA FILE=HCAPLUS ABB=ON ?MITOCHONDRI?
L84	330	SEA FILE=HCAPLUS ABB=ON L65 AND L83
L85	5	SEA FILE=HCAPLUS ABB=ON L84 AND (L26 OR L31 OR L38 OR L40 OR L42 OR L43 OR L46 OR L48 OR L58)

L107 31 L67 OR L85

This search ~~done~~ not done in any other file because Registry answer set was so large. It costs 2¢/Reg# to search non-CA files

=> d ibib abs hitstr l107 1-31; fil hom

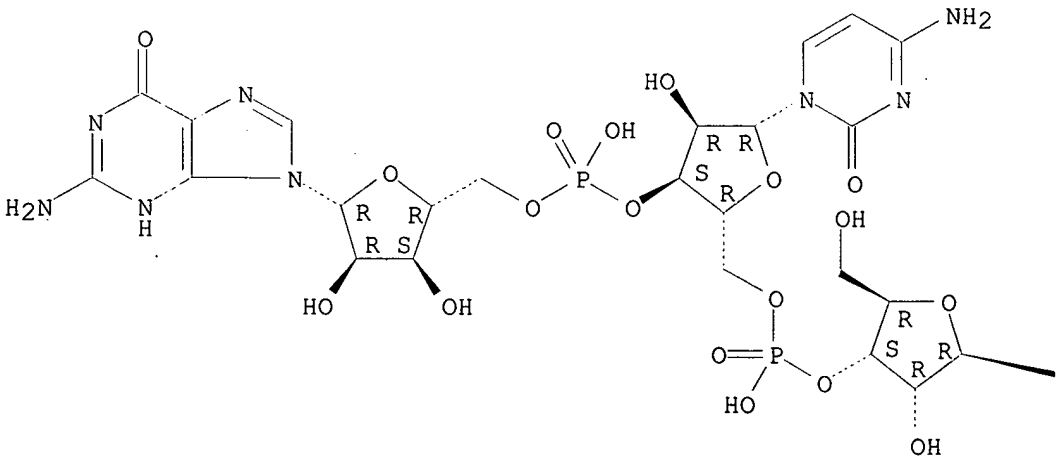
L107 ANSWER 1 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:764661 HCAPLUS
DOCUMENT NUMBER: 133:15274
TITLE: Trinucleotide repeat expansion and Neuropsychiatric disease
AUTHOR(S): Margolis, Russell L.; McInnis, Melvin G.; Rosenblatt, Adam; Ross, Christopher A.
CORPORATE SOURCE: Department of Psychiatry and Behavioral Sciences, Divisions of Neurobiology, Johns Hopkins University School of Medicine, Baltimore, MD, USA
SOURCE: Archives of General Psychiatry (1999), 56(11), 1019-1031
CODEN: ARGPAQ; ISSN: 0003-990X
PUBLISHER: American Medical Association
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 211 refs. Trinucleotide, or triplet, repeats consist of 3 nucleotides consecutively repeated (eg, CCG CCG CCG CCG CCG) within a region of DNA, a not uncommon motif in the genome of humans and other species. In 1991, a new type of genetic mutation was discovered, known as a dynamic or expansion mutation, in which the no. of triplets in a repeat increases and the length becomes unstable. During the past decade, nearly 20 diseases-including Huntington disease, 2 forms of the fragile X syndrome, and myotonic dystrophy-caused by trinucleotide repeat expansions have been identified. The unstable nature of the expanded repeat leads to remarkable patterns of inheritance in these diseases, distinctly at odds with traditional notions of Mendelian genetics. We review the clin. and genetic features of these disorders, with a particular emphasis on their psychiatric manifestations. We also critically examine the hypothesis that expansion mutations may have an etiol. role in psychiatric diseases such as bipolar disorder, schizophrenia, and **autism**.

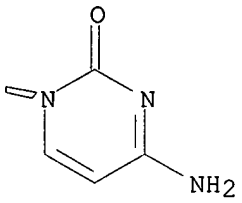
IT **3960-32-5**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(trinucleotide repeat mutation expansion and its assocn. with genetic and psychiatric disorders in human)
RN 3960-32-5 HCAPLUS
CN Guanosine, cytidylyl-(3'.fwdarw.5')-cytidylyl-(3'.fwdarw.5')- (7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



REFERENCE COUNT: 212 THERE ARE 212 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L107 ANSWER 2 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:453397 HCAPLUS
DOCUMENT NUMBER: 131:281217
TITLE: Deletions in the mitochondrial DNA and decrease in the oxidative phosphorylation activity of children with Fanconi syndrome secondary to antiblastic therapy
AUTHOR(S): Di Cataldo, Andrea; Palumbo, Maddalena; Pittala, Donatella; Renis, Marcella; Schiliro, Gino; Russo, Alessandra; Ragusa, Rosalia; Mollica, Florindo; Li Volti, Salvatore
CORPORATE SOURCE: Departments of Pediatric Hematology-Oncology, Biochemistry, and Pediatrics, University of Catania, Italy
SOURCE: Am. J. Kidney Dis. (1999), 34(1), 98-106
CODEN: AJKDDP; ISSN: 0272-6386
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The aim of this study is to verify whether there are deletions in mitochondrial DNA (mtDNA) and disorders in oxidative phosphorylation (Ox-phos) complexes in the pathogenesis of secondary Fanconi syndrome (FS). The authors studied 18 children with tumors who were previously treated with chemotherapy and were off therapy for at least 1 yr. All the children had normal renal function at diagnosis. Only 4 children received ifosfamide (IFO) and platinum compds. The authors evaluated renal function, Ox-phos activity measured on platelets, and mtDNA extd. from platelets for all patients. Only 2 patients, both treated with IFO and carboplatinum (CARBO) for Wilms' tumor and germ-cell tumor, resp., developed FS 1 and 3 yr after termination of therapy. They had decreased activities of Ox-phos that were statistically significant only for NAD-reduced cytochrome-c reductase and cytochrome-c oxidase and specific and unidentified deletions in mtDNA that were not maternally inherited. Therefore, treatment with IFO and CARBO might be responsible for deletions in mtDNA, decreased activity of Ox-phos, and impaired rates of transport

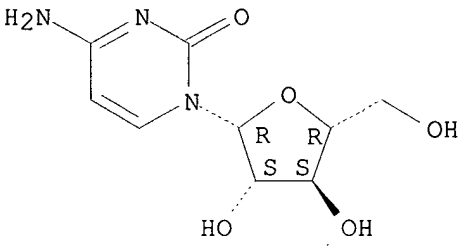
of D-glucose, phosphate, and amino acids.

IT **147-94-4**, Cytarabine
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(deletions in mitochondrial DNA and decreases in oxidative phosphorylation activity in children with Fanconi syndrome secondary to antiblastic therapy)

RN 147-94-4 HCAPLUS

CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L107 ANSWER 3 OF 31 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:450001 HCAPLUS
DOCUMENT NUMBER: 132:47149
TITLE: Investigation of Possible Participation of Nucleoside Transport Systems in the Postischemic Release of Purines and Pyrimidines from Cold Stored Liver
AUTHOR(S): Toshchakov, Vladimir Yu.; Bashkina, Ludmila V.; Shumakov, Valery I.
CORPORATE SOURCE: Institute for Transplantation and Artificial Organs, Moscow, 123182, Russia
SOURCE: Cryobiology (1999), 38(4), 261-272
CODEN: CRYBAS; ISSN: 0011-2240
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

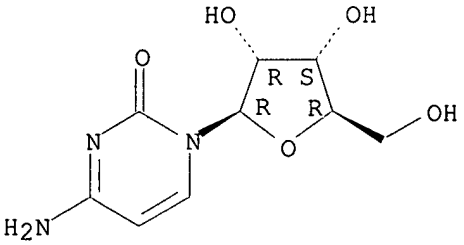
AB The aim of the study was to elucidate the role of nucleoside transport systems in the postischemic release of nucleosides and nucleobases accumulated by the rat liver during cold storage. Livers were preserved for 24 h in Euro-Collins (EC) or in a lactobionate-based soln. (LBS) without exogenous adenosine. The rates of release of uric acid, xanthine, hypoxanthine, inosine, adenosine, uridine, and cytidine were monitored during early reperfusion. The greater part of the purines and pyrimidines (up to 80%) was lost in the first 2 min of reperfusion. After storage in EC, uric acid and xanthine formed more than 90% of the total purines released; nucleosides did not exceed 5% of the total. After storage in LBS, hypoxanthine formed more than 80% of purine efflux and the release of inosine and uridine was increased 5-10 times. These changes were shown to be due to the presence of allopurinol in LBS. Dipyridamole (an inhibitor of equilibrative nucleoside transporters) decreased the efflux of uric acid after storage in EC but residual release remained high. Dipyridamole exerted the most pronounced effect on the release of nucleosides (inosine and uridine) from livers stored in LBS. The use of sodium-free media for liver preservation and reperfusion did not alter the rates of purine and pyrimidine release. We conclude that equilibrative nucleoside transporters mediate the postischemic release of nucleosides and also, but to a less degree, of uric acid. Simple diffusion is an important factor

in the release of nucleobases. Active Na+/nucleoside cotransport does not play an important role in early reperfusion. (c) 1999 Academic Press.

IT **65-46-3**, Cytidine
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(nucleoside transport systems in postischemic release of purines and pyrimidines from cold stored liver)

RN 65-46-3 HCAPLUS
CN Cytidine (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L107 ANSWER 4 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:349890 HCAPLUS
DOCUMENT NUMBER: 131:139091
TITLE: Boswellic acids and malignant glioma: induction of apoptosis but no modulation of drug sensitivity
AUTHOR(S): Glaser, T.; Winter, S.; Groscurth, P.; Safayhi, H.; Sailer, E.-R.; Ammon, H. P. T.; Schabet, M.; Weller, M.
CORPORATE SOURCE: Laboratory of Molecular Neuro-Oncology, Department of Neurology, Institute of Pharmaceutical Sciences, University of Tubingen, Tubingen, 72076, Germany
SOURCE: Br. J. Cancer (1999), 80(5/6), 756-765
CODEN: BJCAAI; ISSN: 0007-0920
PUBLISHER: Churchill Livingstone
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Steroids are essential for the control of **edema** in human malignant glioma patients but may interfere with the efficacy of chemotherapy. Boswellic acids are phytotherapeutic anti-inflammatory agents that may be alternative drugs to corticosteroids in the treatment of **cerebral edema**. Here, the authors report that boswellic acids are cytotoxic to malignant glioma cells at low micromolar concns. In-situ DNA end labeling and electron microscopy reveal that boswellic acids induce apoptosis. Boswellic acid-induced apoptosis requires protein, but not RNA synthesis, and is neither assocd. with free radical formation nor blocked by free radical scavengers. The levels of BAX and BCL-2 proteins remain unaltered during boswellic acid-induced apoptosis. P21 expression is induced by boswellic acids via a p53-independent pathway. Ectopic expression of wild-type p53 also induces p21, and facilitates boswellic acid-induced apoptosis. However, targeted disruption of the p21 genes in colon carcinoma cells enhances rather than decreases boswellic acid toxicity. Ectopic expression of neither BCL-2 nor the caspase inhibitor, CRM-A, is protective. In contrast to steroids, subtoxic concns. of boswellic acids do not interfere with cancer drug toxicity of glioma cells in acute cytotoxicity or clonogenic cell death assays. Also, in contrast to steroids, boswellic acids synergize with the cytotoxic cytokine, CD95 ligand, in inducing glioma cell apoptosis. This effect is probably mediated by inhibition of RNA synthesis and is not

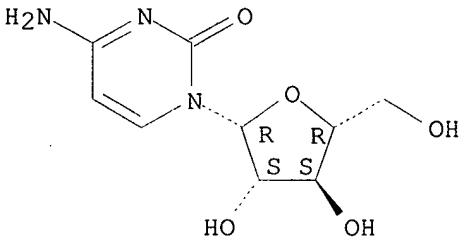
assocd. with changes of CD95 expression at the cell surface. Further studies in lab. animals and in human patients are required to det. whether boswellic acids may be a useful adjunct to the medical management of human malignant glioma.

IT **147-94-4**, Cytarabine
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(boswellic acids and malignant glioma and induction of apoptosis but no modulation of drug sensitivity in relation to mechanism)

RN 147-94-4 HCAPLUS

CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



Proviso edout

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L107 ANSWER 5 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:542765 HCAPLUS
DOCUMENT NUMBER: 129:170542
TITLE: Treatment with dithiocarbamates for atherosclerosis and other cardiovascular and inflammatory diseases by inhibition of expression of VCAM-1
INVENTOR(S): Medford, Russell M.; Alexander, R. Wayne; Offermann, Margaret K.
PATENT ASSIGNEE(S): Emory University, USA
SOURCE: U.S., 17 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5792787	A	19980811	US 1995-486239	19950607

OTHER SOURCE(S): MARPAT 129:170542

AB Dithiocarboxylates, and in particular, dithiocarbamates, block the induced expression of the endothelial cell surface adhesion mol. VCAM-1, and are therefor useful in the treatment of cardiovascular disease, including atherosclerosis, post-angioplasty restenosis, coronary artery diseases, and **angina**, as well as noncardiovascular inflammatory diseases that are mediated by VCAM-1.

IT **24939-03-5**, Poly(I:C)
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(dithiocarbamates for treatment of atherosclerosis and other cardiovascular and inflammatory diseases by inhibition of expression of VCAM-1)

RN 24939-03-5 HCAPLUS

CN 5'-Inosinic acid, homopolymer, complex with 5'-cytidylic acid homopolymer

(1:1) (9CI) (CA INDEX NAME)

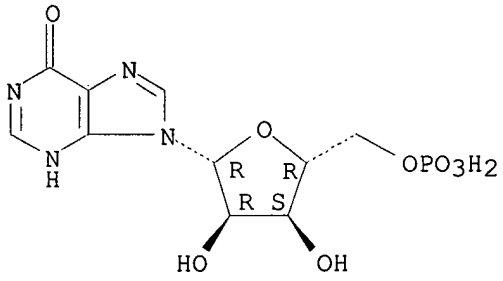
CM 1

CRN 30918-54-8
CMF (C10 H13 N4 O8 P)x
CCI PMS

CM 2

CRN 131-99-7
CMF C10 H13 N4 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.



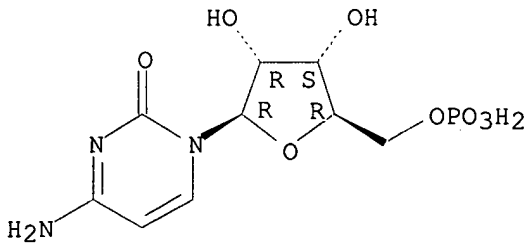
CM 3

CRN 30811-80-4
CMF (C9 H14 N3 O8 P)x
CCI PMS

CM 4

CRN 63-37-6
CMF C9 H14 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.



L107 ANSWER 6 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:353383 HCAPLUS
DOCUMENT NUMBER: 129:132962
TITLE: Nucleotide and Nucleoside Analogs as Inhibitors of Cytosolic 5'-Nucleotidase I from Heart
AUTHOR(S): Garvey, Edward P.; Lowen, Gregory T.; Almond, Merrick R.
CORPORATE SOURCE: Divisions of Biochemistry, Glaxo Wellcome, Research

Searched by Barb O'Bryen, STIC 308-4291

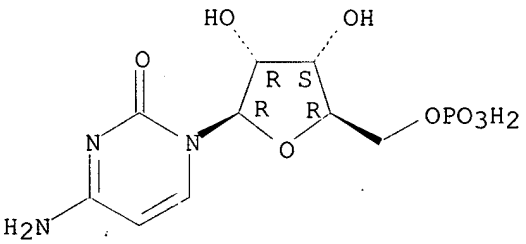
Triangle Park, NC, 27709, USA
SOURCE: Biochemistry (1998), 37(25), 9043-9051
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Substrate and product specificity studies were used to develop inhibitors of the cytosolic 5'-nucleotidase I (c-N-I) from myocardium. As measured by Vmax/Km, c-N-I preferred pyrimidine 2'-deoxyribonucleotides as substrates with thymidine monophosphate (TMP) being the most efficient. In product inhibition studies, thymidine inhibited noncompetitively and inorg. phosphate inhibited competitively, consistent with an ordered release of nucleoside prior to phosphate. Mirroring nucleotide substrate specificities, pyrimidine nucleosides were more potent product inhibitors than purine nucleosides. Thus, pyrimidine nucleotide and nucleoside analogs were developed as inhibitors. Phosphonate analogs of TMP were synthesized by a novel method. The most potent was the 5'-phosphonate of 3'-deoxythymidine (ddT) (apparent Ki value of 63 nM). In addn., pyrimidine nucleoside analogs were inhibitors with 5-ethynyl-2',3'-dideoxyuridine being the most potent (apparent Ki value of 3.7 .mu.M). The most potent nucleotide and nucleoside inhibitor were both greater than 1000-fold more potent inhibiting c-N-I than the cytosolic 5'-nucleotidase II. The nucleoside analog was also greater than 1000-fold more potent against c-N-I than the membrane ecto-5'-nucleotidase (e-N). Because the phosphonate analogs measurably inhibited e-N (apparent Ki values of 6-12 .mu.M), the selectivity of the phosphonates for c-N-I vs. e-N was less (40-200-fold). Because of the high selectivity for c-N-I vs. both of the other 5'-nucleotidases, the nucleoside inhibitors of c-N-I may be useful biochem. tools in discerning the role that c-N-I plays in generating adenosine within myocardium.

IT **63-37-6**, 5'-Cmp
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(nucleotide and nucleoside analogs as inhibitors of cytosolic 5'-nucleotidase I from heart)

RN 63-37-6 HCAPLUS
CN 5'-Cytidylic acid (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 7 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:714034 HCAPLUS
DOCUMENT NUMBER: 128:881
TITLE: Molecular and cytogenetic investigations of the fragile X region including the Frax A and Frax E CGG trinucleotide repeat sequences in families multiplex for **autism** and related phenotypes
AUTHOR(S): Gurling, H. M. D.; Bolton, P. F.; Vincent, J.; Melmer, G.; Rutter, M.
CORPORATE SOURCE: Molecular Psychiatry Laboratory, Department of Psychiatry, University College London Medical School,

London, W1P 6DB, UK
SOURCE: Hum. Hered. (1997), 47(5), 254-262
CODEN: HUHEAS; ISSN: 0001-5652
PUBLISHER: Karger
DOCUMENT TYPE: Journal
LANGUAGE: English

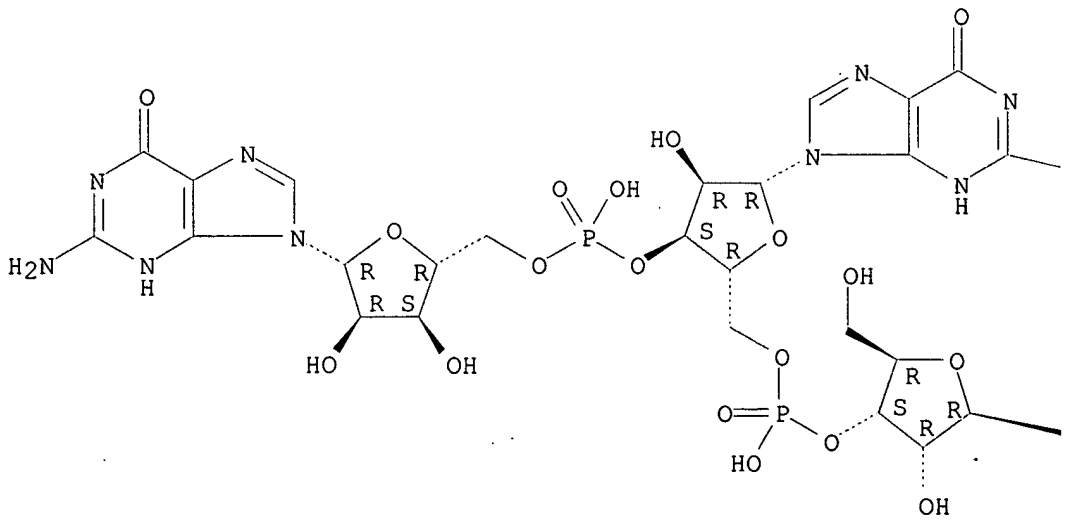
AB We undertook mol. and cytogenetic analyses in 25 families multiplex for **autism** and related disorders. Three of the multiplex families exhibited fragile X, and the affected off-spring all exhibited CGG triplet repeat insertion mutations in the FMR-1 gene. One of these families contained an affected pair of monozygotic female twins. Both had similar-sized CGG triplet repeat expansions, but different phenotypic manifestations. One suffered from **autism** and the other from mild mental retardation and marked social anxiety. PCR and Southern hybridization anal. of the CGG repeat sequences characterizing fragile X A (Frax A) and E and the methylation status of FMR-1 showed no evidence of abnormal CGG repeat expansion or FMR-1 hypermethylation in the remaining 22 multiplex families. Moreover, there was no correlation between the Frax A or E (CGG)n repeat length with affected status, nor any assocn. with the low-level (<3%) expression of cytogenetic fragility at Xq27 previously reported in these families. Our findings indicate that most instances of recurrence in families multiplex for **autism** and related disorders are not accounted for by Frax A and E. They also indicate that the phenotypic manifestations of Frax A may be influenced by stochastic, environmental and other biol. factors.

IT **5875-29-6**
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(trinucleotide repeat; mol. and cytogenetic investigations of the fragile X region including the Frax A and Frax E CGG trinucleotide repeat sequences in families multiplex for **autism** and related phenotypes)

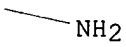
RN 5875-29-6 HCAPLUS
CN Guanosine, cytidylyl-(3'.fwdarw.5')-guanylyl-(3'.fwdarw.5')- (7CI, 8CI, 9CI) (CA INDEX NAME)

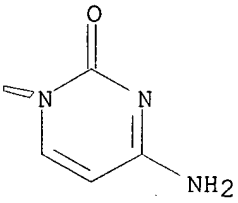
Absolute stereochemistry.

PAGE 1-A



PAGE 1-B





L107 ANSWER 8 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:431442 HCAPLUS
DOCUMENT NUMBER: 125:76393
TITLE: Use of GABA agonists in the treatment of emesis
INVENTOR(S): Bays, David Edmund; Bountra, Charanjit
PATENT ASSIGNEE(S): Glaxo Group Limited, UK
SOURCE: PCT Int. Appl., 9 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9611680	A2	19960425	WO 1995-EP4025	19951012
WO 9611680	A3	19960627		
W: AL, AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ				
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9537458	A1	19960506	AU 1995-37458	19951012

PRIORITY APPLN. INFO.: GB 1994-20784 19941014
WO 1995-EP4025 19951012

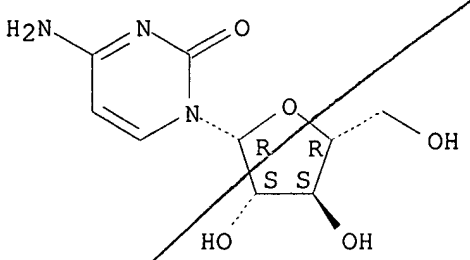
AB Selected GABA agonists having an agonist action at GABAB receptors are used for the treatment of emesis. Specific compds., as well as compds. from other patents, are claimed. In a cisplatin emesis model, (.+-.)-Baclofen inhibited emesis at 1.0 mg/kg s.c.

IT **147-94-4**, Cytarabine
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(emesis from; GABA agonists for emesis treatment)

RN 147-94-4 HCAPLUS

CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 9 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:287043 HCAPLUS
DOCUMENT NUMBER: 124:339965
TITLE: cDNA cloning of a human homolog of the Caenorhabditis elegans cell fate-determining gene mab-21: expression, chromosomal localization and analysis of a highly polymorphic (CAG)n trinucleotide repeat
AUTHOR(S): Margolis, Russell L.; Stine, O. Colin; McInnis, Melvin G.; Ranen, Neal G.; Rubinsztein, David C.; Leggo, Jayne; Brando, Lorraine V. Jones; Kidwai, Arif S.; Loev, Scott J.; et al.
CORPORATE SOURCE: Laboratory Molecular Neurobiology, Johns Hopkins University School Medicine, Baltimore, MD, 21205-2196, USA
SOURCE: Hum. Mol. Genet. (1996), 5(5), 607-616
CODEN: HMGEE5; ISSN: 0964-6906
DOCUMENT TYPE: Journal
LANGUAGE: English

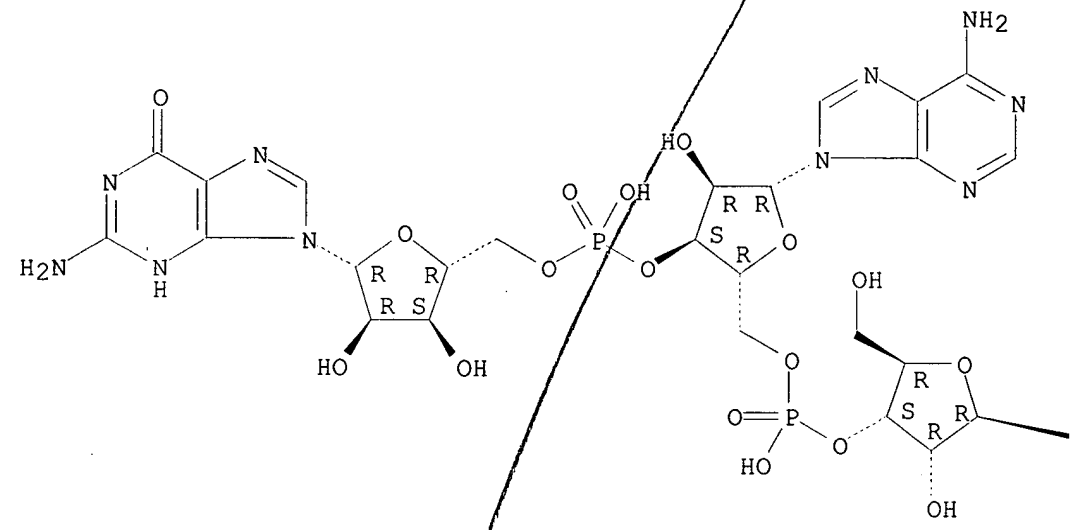
AB The two most consistent features of the diseases caused by trinucleotide repeat expansion-neuropsychiatric symptoms and the phenomenon of genetic anticipation-may be present in forms of dementia, hereditary ataxia, Parkinsonism, bipolar affective disorder, schizophrenia and **autism**. To identify candidate genes for these disorders, we have screened human brain cDNA libraries for the presence of gene fragments contg. polymorphic trinucleotide repeats. Here we report the cDNA cloning of CAGR1, originally detected in a retinal cDNA library. The 2743 bp cDNA contains a 1077 bp open reading frame encoding 359 amino acids. This amino acid sequence is homologous (56% amino acid identity and 81% amino acid conservation) to the Caenorhabditis elegans cell fate-detg. protein mab-21. CAGR1 is expressed in several human tissues, most prominently in the cerebellum, as a message of .apprx.3.0 kb. The gene was mapped to 13q13, just telomeric to D13S220. A 5'-untranslated CAG trinucleotide repeat is highly polymorphic, with repeat length ranging from six to 31 triplets and a heterozygosity of 87-88% in 684 chromosomes from several human populations. One allele from an individual with an atypical movement disorder and bipolar affective disorder type II contains 46 triplets, 15 triplets longer than any other allele detected. Though insufficient data are available to link the long repeat to this clin. phenotype, an expansion mutation of the CAGR1 repeat can be considered a candidate for the etiol. of disorders with anticipation or developmental abnormalities, and particularly any such disorders linked to chromosome 13.

IT **4353-69-9**
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cDNA cloning of a human homolog of Caenorhabditis elegans cell fate-detg. gene mab-21: expression, chromosomal localization and anal. of a highly polymorphic (CAG)n trinucleotide repeat)
RN 4353-69-9 HCAPLUS

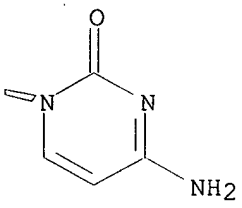
CN Guanosine, cytidylyl-(3'.fwdarw.5')-adenylyl-(3'.fwdarw.5')- (7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



L107 ANSWER 10 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:285838 HCAPLUS
DOCUMENT NUMBER: 122:46504
TITLE: GABA agonists for the treatment of emesis
INVENTOR(S): Bays, David Edmund; Bountra, Charanjit
PATENT ASSIGNEE(S): Glaxo Group Ltd., UK
SOURCE: PCT Int. Appl., 12 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

Searched by Barb O'Bryen, STIC 308-4291

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9425016	A1	19941110	WO 1994-EP1319	19940421
W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KG, KP, KR, KZ, LK, LU, LV, MD, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9467221	A1	19941121	AU 1994-67221	19940421
EP 695180	A1	19960207	EP 1994-915548	19940421
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 08509238	T2	19961001	JP 1994-523876	19940421
US 5719185	A	19980217	US 1995-532813	19951023

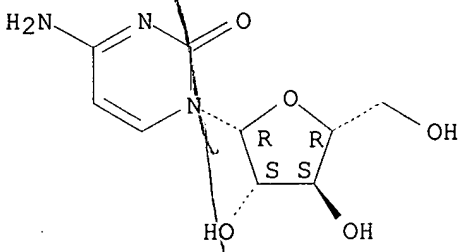
PRIORITY APPLN. INFO.: GB 1993-8430 19930423
WO 1994-EP1319 19940421

AB The present invention relates to the use of GABA agonists having an agonist action at GABA .beta.-receptors in the treatment of emesis. For example, s.c. administration of (-)-baclofen at 1 mg/kg to ferrets immediately after whole body irradn. inhibited the emesis detd. by comparison with appropriate controls.

IT **147-94-4**, Cytarabine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(vomiting from; GABA agonists for treatment of emesis)

RN 147-94-4 HCAPLUS
CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

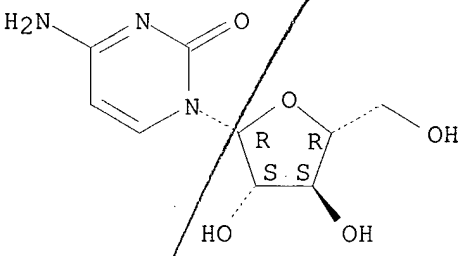


L107 ANSWER 11 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:621979 HCAPLUS
DOCUMENT NUMBER: 121:221979
TITLE: Virucides for the treatment of a group of related disorders
INVENTOR(S): Horrobin, David Frederick; Bond, Peter
PATENT ASSIGNEE(S): Scotia Holdings PLC, UK
SOURCE: Eur. Pat. Appl., 6 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 615750	A2	19940921	EP 1994-301106	19940216
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				

AU 9455186 A1 19940825 AU 1994-55186 19940217
PRIORITY APPLN. INFO.: GB 1993-3157 19930217
AB A drug selected from the group consisting of acyclovir, gancyclovir, famcyclovir, AZT, anhydrothymidine D 4T, ddC, ddA, cytosine arabinoside, adenosine arabinoside, and derivs. thereof is used for the treatment of one or more of the following conditions when a herpes virus, particularly herpes simplex virus, is involved in the causation thereof: depression, **irritable bowel** syndrome, fibromyalgia, headaches, anxiety, panic disorder, narcolepsy, Tourette's disorder, kleptomania, bulimia nervosa, obsessive compulsive disorder, **attention deficit** disorder with hyperactivity, cataplexy, psychopathic disorder, gingivitis, and dental caries.
IT **147-94-4**, Cytosine arabinoside
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(virucides for treatment of herpes simplex virus infection symptoms)
RN 147-94-4 HCAPLUS
CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 12 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:440411 HCAPLUS
DOCUMENT NUMBER: 117:40411
TITLE: thiolated oligo- and polynucleotides for treating HIV infections
INVENTOR(S): Bardos, Thomas J.; Ho, Yau Kwan; Aradi, Janos; Schinazi, Raymond F.
PATENT ASSIGNEE(S): State University of New York, Albany, USA
SOURCE: PCT Int. Appl., 42 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9203127	A1	19920305	WO 1991-US5919	19910815
W: AT, AU, BB, BG, BR, CA, CH, CS, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MC, MG, MN, MW, NL, NO, PL, RO, SD, SE, SU, US				
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GN, GR, IT, LU, ML, MR, NL, SE, SN, TD, TG				
AU 9184971	A1	19920317	AU 1991-84971	19910815

PRIORITY APPLN. INFO.: US 1990-568131 19900816
WO 1991-US5919 19910815
AB The title compds. are therapeutically effective for inhibiting HIV-1 infections and for treating AIDS. The nucleotides used are 5-mercaptopoly(C), 5-mercaptopoly(dC), 5-mercaptopoly(U), and 5-mercaptopoly(dU) contg. 2-30% thiolation, the corresponding 4 oligonucleotides contg. 3-10% thiolation, or a regional sense or

anti-sense 5-thiolated oligonucleotide corresponding to at least a portion of a primer tRNA (esp. tRNALys) of HIV reverse transcriptase. Thus, poly[U91,[5-mercapto-(U)]9] showed a 50% inhibitory concn. of 9 .mu.M against HIV-1 in infected human lymphocytes in vitro as evaluated morphol., by indirect immunofluorescence, and by reverse transcriptase activity. The thiolated oligo- and polynucleotides were prepd. by chem. or enzymic synthesis or by partial thiolation with **NaSH** of partial alk. hydrolyzates of poly(C) or poly(U).

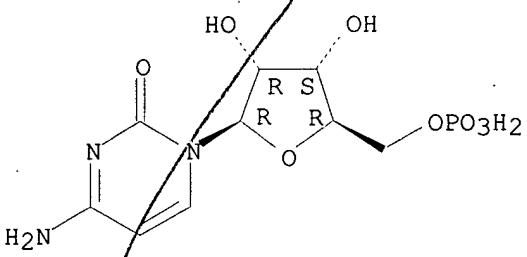
IT **30811-80-4D**, Polycytidylic acid, 5-thiolated
RL: BIOL (Biological study)
(human immunodeficiency virus inhibition with)

RN 30811-80-4 HCAPLUS
CN 5'-Cytidylic acid, homopolymer (9CI) (CA INDEX NAME)

CM 1

CRN 63-37-6
CMF C9 H14 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.



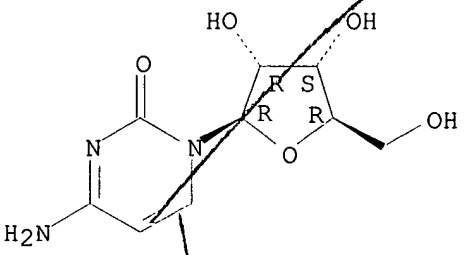
L107 ANSWER 13 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:653232 HCAPLUS
DOCUMENT NUMBER: 115:253232
TITLE: Pyrimidine nucleotide synthesis in the rat kidney in early **diabetes**
AUTHOR(S): Kunjara, Sirilaksana; Sochor, Milena; Ali, Murad; Drake, Adrian; Greenbaum, Leslie; McLean, Patricia
CORPORATE SOURCE: Dep. Biochem., Univ. Coll., London, W1P 6DB, UK
SOURCE: Biochem. Med. Metab. Biol. (1991), 46(2), 215-25
CODEN: BMMBES; ISSN: 0885-4505
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Early renal hypertrophy of **diabetes** is assocd. with increases in the tissue content of RNA, DNA, and sugar nucleotides involved in the formation of carbohydrate-contg. macromols. The authors have previously reported an increase in the activity of enzymes of the de novo and salvage pathways of purine synthesis in early **diabetes**; the present communication explores the changes in the pathways of pyrimidine synthesis. Measurements have been made of key enzymes of the de novo and salvage pathways at 3, 5, and 14 days after induction of **diabetes** with streptozotocin (STZ), phosphoribosyl pyrophosphate (PPRibP), and some purine and pyrimidine bases. Carbamoyl-phosphate synthetase II, the rate-limiting enzyme of the de novo route, did not increase in the first 5 days after STZ treatment, the period of most rapid renal growth; a rise was seen at 14 days (+38%). Dihydroorotate dehydrogenase, a **mitochondrial** enzyme, showed the most marked rise (+147%) at 14 days. The conversion of orotate to UMP, catalyzed by the enzymes of complex II, was increased at 3 days (+42%), a rise sustained to 14 days. The salvage route enzyme, uracil phosphoribosyltransferase (UPRTase),

showed a pattern of change similar to complex II. The effect of the decreased concn. of PPRibP on the activities of CPSII, for which it is an allosteric activator, and on activities of OPRTase and UPRTase, for which it is an essential substrate, is discussed with respect to the relative Ka and Km values for PPRibP and the possibility of metabolite channeling.

IT **65-46-3**, Cytidine
RL: BIOL (Biological study)
(of kidney, in renal hypertrophy in **diabetes** mellitus)
RN 65-46-3 HCAPLUS
CN Cytidine (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



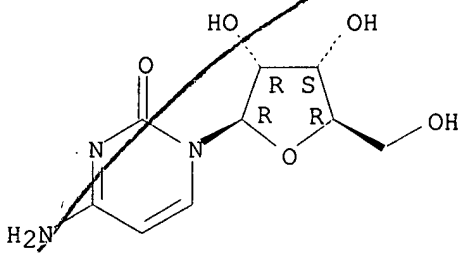
L107 ANSWER 14 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:244644 HCAPLUS
DOCUMENT NUMBER: 114:244644
TITLE: Comparison of nucleoside concentrations in blood of fish with and without tumors
AUTHOR(S): Kuehl, Douglas W.; Eisenschenk, Linda; Naumann, Sandra; Johnson, Rodney D.; Regal, Ronald; Barnidge, Phyllis; McKim, James, Jr.
CORPORATE SOURCE: Environ. Res. Lab., U.S. EPA, Duluth, MN, 55804, USA
SOURCE: Bull. Environ. Contam. Toxicol. (1991), 46(5), 713-19
CODEN: BECTA6; ISSN: 0007-4861
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Blood samples were used to establish diagnostic parameters for control rainbow trout (Oncorhynchus mykiss) and those with tumors induced by aflatoxins. A 2nd set of blood samples was from field collected back bullheads (Ictalurus **melas**). Inosine was the major nucleoside in all of the rainbow trout samples. Guanosine was most often the nucleoside at the 2nd highest concn. in the trout samples; however, the concn. of xanthosine exceeded guanosine in 2 of the toxicant-exposed fish. The av. concn. of guanosine was 11% that of inosine in the trout. Cytidine, pseudouridine, and uridine were also identified as minor nucleosides. The same 6 nucleosides were identified in bullhead samples, and again inosine was identified as the major nucleoside in most of the samples. The concn. of guanosine exceeded inosine in 3 bullhead samples. The concns. of inosine and guanosine in trout were bimodal with values <380 .mu.g/mL and >800 .mu.g/mL, and <35 .mu.g/mL and >75 .mu.g/mL for each compd., resp. The fish in the high range of concns. were the same for each compd., and were female, indicating a concn. bias may possibly be influenced by the sex of the fish. A similar bimodal distribution was obsd. in the bullhead data, where concns. <175 .mu.g/mL and >300 .mu.g/mL, and <80 .mu.g/mL and >195 .mu.g/mL for inosine and guanosine, resp., were measured. Trout with tumors could not be distinguished from trout without tumors by comparing the concn. of any individual nucleoside.

IT **65-46-3**, Cytidine
RL: BIOL (Biological study)
(of blood serum, of fish, tumor effect on)
RN 65-46-3 HCAPLUS

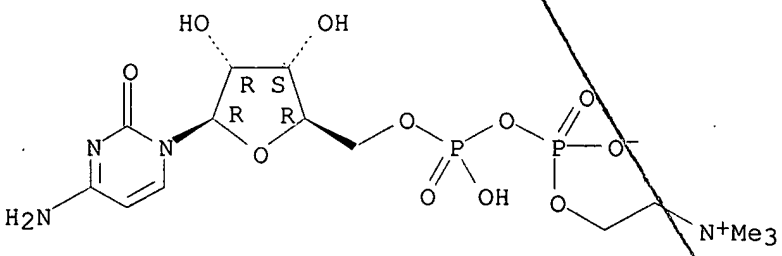
CN Cytidine (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 15 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:602943 HCAPLUS
DOCUMENT NUMBER: 105:202943
TITLE: Protection against experimental acute cerebral ischemia by CDP-choline
AUTHOR(S): Le Poncin-Lafitte, M.; Duterte, D.; Lageron, A.; Rapin, J. R.
CORPORATE SOURCE: Inst. Natl. Rech. Prev. Vieillissement Cereb., Hop. Bicetre, Le Kremlin-Bicetre, F-94270, Fr.
SOURCE: Agressologie (1986), 27(5), 413-16
CODEN: AGSOA6; ISSN: 0002-1148
DOCUMENT TYPE: Journal
LANGUAGE: French
AB In rats with exptl. acute **cerebral** ischemia induced by intracarotid injection of 85Sr-labeled wax or carbon microspheres, i.p. administration of CDP-choline [**987-78-0**] (30 mg/kg) 1, 3, and 5 h after ischemia reduced the degree of vasogenic **edema** in the **cerebral** hemispheres. CDP-choline also protected the blood-brain barrier, as shown by the reduced amt. of 131T escaping from **cerebral** vessels after treatment. Treatment also reduced the severity of microinfarction, as shown by decreased infart-tissue activity of lactate dehydrogenase [9001-60-9], succinate dehydrogenase [9002-02-2], monoamine oxidase [9001-66-5], and acid phosphatase [9001-77-8].
IT **987-78-0**
RL: BIOL (Biological study)
(brain ischemia protection by)
RN 987-78-0 HCAPLUS
CN Cytidine 5'-(trihydrogen diphosphate), P'-[2-(trimethylammonio)ethyl] ester, inner salt (9CI) (CA INDEX NAME)

Absolute stereochemistry.



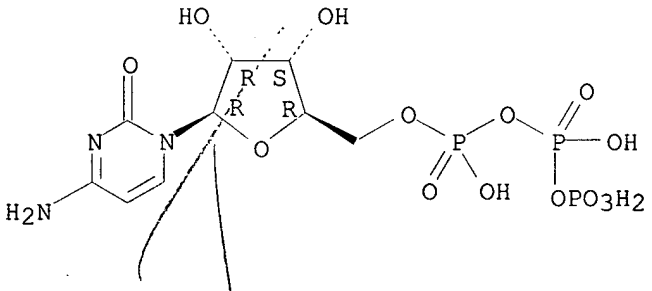
L107 ANSWER 16 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:513120 HCAPLUS

DOCUMENT NUMBER: 105:113120
TITLE: Adenylosuccinase deficiency
AUTHOR(S): Van den Berghe, G.; Jaeken, J.
CORPORATE SOURCE: Int. Inst. Cell. Mol. Pathol., Univ. Louvain Brussels, Brussels, Belg.
SOURCE: Adv. Exp. Med. Biol. (1986), 195A(Purine Pyrimidine Metab. Man 5, Pt. A), 27-33
CODEN: AEMBAP; ISSN: 0065-2598
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In 2 children (brother and sister, patients B and C) with mental retardation and **autism** , adenylosuccinase (E.C. 4.3.2.2) (I) activity was markedly below normal in liver, kidney, muscle, fibroblasts, and lymphocytes; the percent change in I activity in these 2 patients is compared with previous data from another patient (patient A) with the same clin. conditions. Variations in the amts. of 13 nucleotides present in liver, kidney, and muscle of patients A, B, and C are described and contrasted with normal values. Sep. studies were conducted with purified rat liver cytoplasmic 5'-nucleotidase to assess the formation of succinyladenosine (II) and succinylaminoimidazole carboxamide riboside (III) in the I-deficent tissues are presented and their clin. significance discussed. Therapeutic trials with allopurinol (33 mg/kg for 3 wk) and Na benzoate (250 mg/kg for 4 wks) did not modify cerebrospinal fluid or plasma concns. of II and III or the urinary excretions of succinylpurines. Aminoimidazole carboxamide (10 mg/kg for 1/ days and 20 mg/kg for 2/ days) resulfted in a slight improvement in behavior, an .apprx.2-fold increase in uric acid excretion, and no change in succinylpurines excretion.

IT **65-47-4**
RL: BIOL (Biological study)
(of tissues, in adenylosuccinase deficiency in children)
RN 65-47-4 HCAPLUS
CN Cytidine 5'-(tetrahydrogen triphosphate) (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 17 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:116113 HCAPLUS
DOCUMENT NUMBER: 104:116113
TITLE: Lipid nanopellent oral drug formulation
INVENTOR(S): Speiser, Peter
PATENT ASSIGNEE(S): Rentschler, Dr., Arzneimittel G.m.b.H. und Co., Fed. Rep. Ger.
SOURCE: Ger. Offen., 35 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 3421468	A1	19851219	DE 1984-3421468	19840608

Searched by Barb O'Bryen, STIC 308-4291

EP 167825	A2	19860115	EP 1985-106926	19850604
EP 167825	A3	19870121		
EP 167825	B1	19900808		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
AT 55243	E	19900815	AT 1985-106926	19850604
JP 61056122	A2	19860320	JP 1985-120726	19850605
US 4880634	A	19891114	US 1987-66459	19870626
PRIORITY APPLN. INFO.:			DE 1984-3421468	19840608
			EP 1985-106926	19850604
			US 1985-740771	19850630

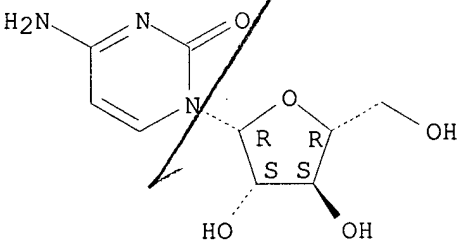
AB Lipid nanopellets (80-800 nm), as aq. colloidal suspensions, are carrier systems for oral drugs. The lipids are satd. fatty acids, their esters with glycerol and with other polyalcs., and fatty alcs. The system contains natural or artificial surfactants. Thus, a mixt. of 2 g tristearin and 0.6 g testosterone undecanoate was melted at 85.degree. and 0.4 g phospholipon 100-H in 4 mL CHCl3 was added. The CHCl3 was evapd. and 0.04 Na cholate in 200 mL water was added, followed by stirring and ultrasonication, to give the nanopellet suspension.

IT **147-94-4**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(lipid nanopellets, for oral administration as aq. colloidal emulsion)

RN 147-94-4 HCAPLUS

CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)

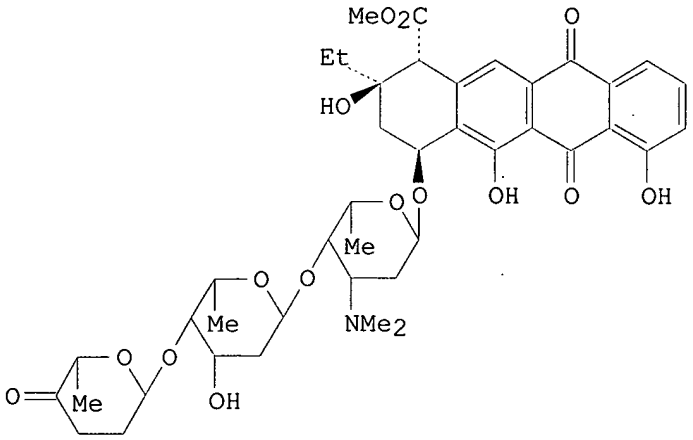
Absolute stereochemistry.



L107 ANSWER 18 OF 31 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER:	1985:481345 HCAPLUS
DOCUMENT NUMBER:	103:81345
TITLE:	Aclarubicin: experimental and clinical experience
AUTHOR(S):	Roethig, H. J.; Kraemer, H. P.; Sedlacek, H. H.
CORPORATE SOURCE:	Res. Lab., Behringwerke A.-G., Marburg/Lahn, D-355, Fed. Rep. Ger.
SOURCE:	Drugs Exp. Clin. Res. (1985), 11(2), 123-5 CODEN: DECRDP; ISSN: 0378-6501
DOCUMENT TYPE:	Journal
LANGUAGE:	English

GI



I

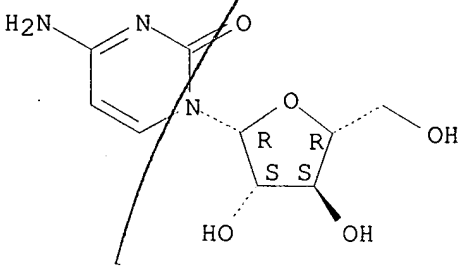
AB The therapeutic index of aclarubicin (I) [57576-44-0] (efficacy related to toxicity) was higher than that of doxorubicin and daunorubicin, using a proper dose schedule. Single dose therapy with aclarubicin showed only marginal efficacy, whereas multiple divided dose therapy exhibited efficacy comparable to that of doxorubicin and daunorubicin. Thus, for clin. trials 2 dose schedules were designed: 25 mg/m2/day, days 1-7, for acute leukemia; and 30 mg/m2/day, days 1-4, for solid tumors. Aclarubicin was highly active in acute leukemia, with 58% complete remissions in patients in 1st relapse of acute myelogenous leukemia. Good results were also seen in acute leukemia in combination with cytosine arabinoside [**147-94-4**] and thioguanine [154-42-7]. In clin. trials with breast cancer and thyroid cancer, the efficacy was in the same range as would be expected for doxorubicin, but side-effects were markedly reduced. Anorexia, mild nausea and infrequent vomiting were obsd. Myelosuppression was common but dose redn. was not necessary. There was no alopecia and no **congestive heart failure**.

IT **147-94-4**
RL: BIOL (Biological study)
(neoplasm inhibition by aclarubicin and thioguanine and, in humans)

RN 147-94-4 HCAPLUS

CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 19 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:508439 HCAPLUS
DOCUMENT NUMBER: 101:108439
TITLE: Effects of neonatal hypoxia on microcephalic mice caused by cytosine arabinoside
AUTHOR(S): Kimura, Shoko; Kameyama, Yoshiro

Searched by Barb O'Bryen, STIC 308-4291

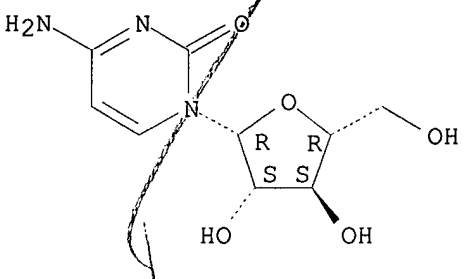
CORPORATE SOURCE: Res. Inst. Environ. Med., Nagoya Univ., Nagoya, Japan
SOURCE: Kankyo Igaku Kenkyusho Nenpo (Nagoya Daigaku) (1984), 35, 254-5
CODEN: NDKIA2; ISSN: 0369-3570
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB Attempts were made to produce a model of **cerebral palsy** by i.p. injecting cytosine arabinoside at 30 mg/kg into pregnant mice and by subjecting the newborns to a hypoxic environment. Cytosine arabinoside induced microcephaly, but the subsequent hypoxia did not induce pathol. effects related to **cerebral palsy**.

IT **147-94-4**
RL: BIOL (Biological study)
(**cerebral palsy** animal model from hypoxia and)

RN 147-94-4 HCAPLUS
CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 20 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:508346 HCAPLUS
DOCUMENT NUMBER: 101:108346
TITLE: Protective role of adenine nucleotide translocase in oxygen-deficient hearts
AUTHOR(S): Pande, Shri V.; Goswami, Tapas; Parvin, Rehana
CORPORATE SOURCE: Lab. Intermediary Metab., Clin. Res. Inst. Montreal, Montreal, PQ, H2W 1R7, Can.
SOURCE: Am. J. Physiol. (1984), 247(1, Pt. 2), H25-H34
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English

AB At subsatg. concns. of palmitoyl-CoA, the carnitine-dependent oxidn. of the palmitoyl portion by uncoupled rat heart **mitochondria** was stimulated by ADP or ATP. This effect was traced to the prevention of acyl-CoA binding to adenine nucleotide translocase and the consequent sparing of acyl-CoA for acylcarnitine formation. Palmitoyl-CoA oxidn. was stimulated by ITP also, although ITP served neither as a transportable substrate nor as an inhibitor of ADP transport. ITP and other nontransportable nucleoside di(tri)phosphates prevented octanoyl-CoA binding to **mitochondria**. ITP was bound to **mitochondria**, and this binding was reversed by ADP, octanoyl-CoA, and carboxyatractyloside. Thus, besides a substrate site, there is a site on the translocase that binds nucleoside di(tri)phosphates, CoA and its esters, and atractylosides; inhibition of the translocase results, however, only from the binding of CoA esters of fatty acids and of atractylosides. It is suggested that in O-deficient hearts, when nucleotides decline and fatty acyl-CoA rises, the binding of the latter to the translocase becomes operational to slow fatty acylcarnitine prodn. By retarding the rise in amphipathic burden, this mechanism could protect heart against irreversible damage during brief periods of ischemia or

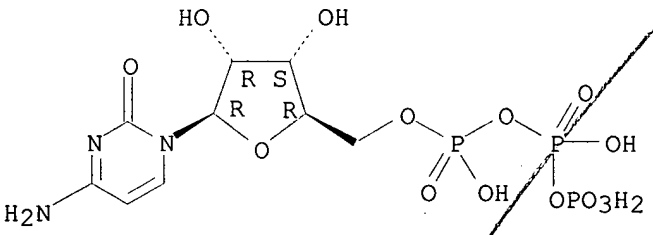
hypoxia.

IT **65-47-4**
RL: BIOL (Biological study)
(octanoyl-CoA binding by **heart mitochondria** response to, **heart ischemia** in relation to)

RN 65-47-4 HCAPLUS

CN Cytidine 5'-(tetrahydrogen triphosphate) (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 21 OF 31 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1984:448396 HCAPLUS

DOCUMENT NUMBER: 101:48396

TITLE: Treatment of vasogenic edema by CDP-choline, a metabolic precursor of phospholipids (experimental and clinical data)

AUTHOR(S): Cohadon, F.; Rigoulet, M.; Averet, N.; Richer, E.

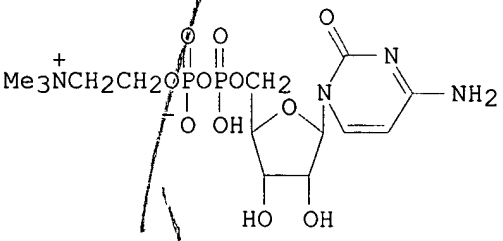
CORPORATE SOURCE: Lab. Neurochir. Exp., Univ. Bordeaux II, Bordeaux, 33076, Fr.

SOURCE: Recent Prog. Study Ther. Brain Edema, [Proc. Int. Symp. Brain Edema], 5th (1984), Meeting Date 1982, 691-700. Editor(s): Go, K. G.; Baethmann, A. Plenum: New York, N. Y.
CODEN: 51TOA5

DOCUMENT TYPE: Conference

LANGUAGE: English

GI



I

AB In rabbits with exptl. brain **edema**, treatment with CDP choline (I) [**987-78-0**] (20 mg/kg, i.v., every 24 h) restored the **mitochondrial** ATPase [9000-83-3] to normal, enhanced Na+-K+-ATPase activity at a high K+/Na+ ratio but not at a low K+/Na+ ratio, and decreased the brain water content. In human patients suffering from a diffuse brain insult (**cerebral** contusions and/or severe concussion), administration of I during the acute phase after the injury at a time brain **edema** is likely to occur shortened the period of time with severe disorders of consciousness and accelerated the restoration of neurol. deficits. I may act by limiting the propagation of brain **edema** or enhancing the resoln. of **edema** fluid. However, I did not affect the permanent deficits of neurol. function

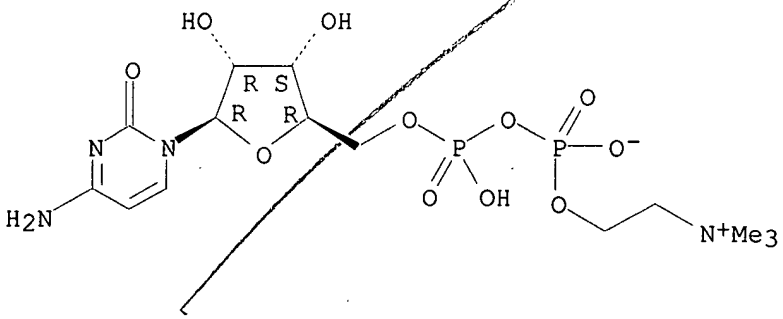
resulting from the brain injury.

IT **987-78-0**
RL: BIOL (Biological study)
(**brain** edema and **injury** treatment with, in humans and lab. animals)

RN 987-78-0 HCAPLUS

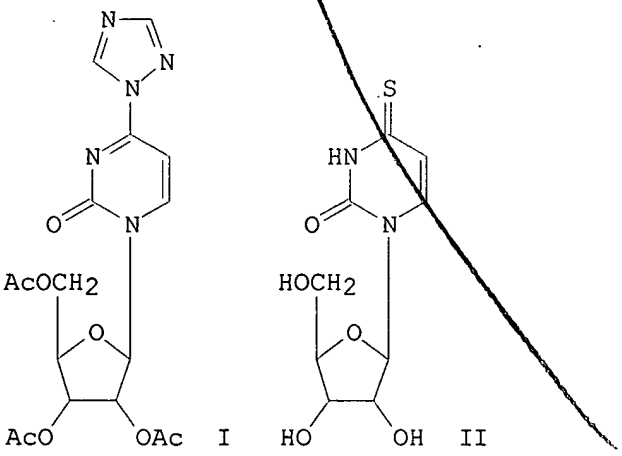
CN Cytidine 5'-(trihydrogen diphosphate), P'-[2-(trimethylammonio)ethyl] ester, inner salt (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 22 OF 31 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1982:527988 HCAPLUS
DOCUMENT NUMBER: 97:127988
TITLE: Chemical conversion of uridine into 4-thiouridine via the 4-(1,2,4-triazol-1-yl)pyrimidin-2(1H)-one intermediate
AUTHOR(S): Sung, Wing L.
CORPORATE SOURCE: Div. Biol. Sci., Natl. Res. Counc. Canada, Ottawa, K1A 0R6, Can.
SOURCE: J. Chem. Soc., Chem. Commun. (1982), (9), 522-3
CODEN: JCCCAT; ISSN: 0022-4936
DOCUMENT TYPE: Journal
LANGUAGE: English
GI

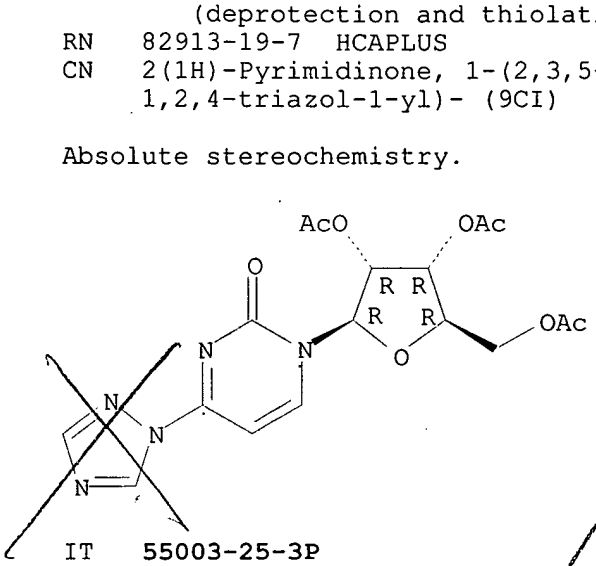


AB Treatment of the uridine-derived triazole I with **NaSH** in Me2CO-H2O for 15 min gave 85% 2',3',5'-tri-O-acetyl-4-thiouridine which on deacetylation gave 89% thiouridine (II).

IT **82913-19-7**
RL: RCT (Reactant)

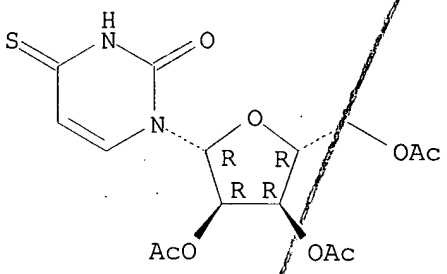
(deprotection and thiolation of)
RN 82913-19-7 HCAPLUS
CN 2(1H)-Pyrimidinone, 1-(2,3,5-tri-O-acetyl-.beta.-D-ribofuranosyl)-4-(1H-1,2,4-triazol-1-yl)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



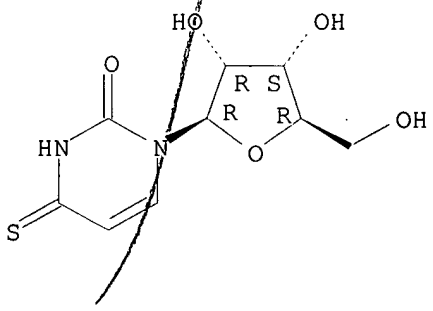
IT **55003-25-3P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and deacetylation of)
RN 55003-25-3 HCAPLUS
CN Uridine, 4-thio-, 2',3',5'-triacetate (7CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



IT **13957-31-8P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of, from triazolyl(triacetylribofuranosyl)pyrimidinone)
RN 13957-31-8 HCAPLUS
CN Uridine, 4-thio- (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 23 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:215425 HCAPLUS
DOCUMENT NUMBER: 96:215425
TITLE: Prolonged myocardial nucleotide depletion after brief ischemia in the open-chest dog

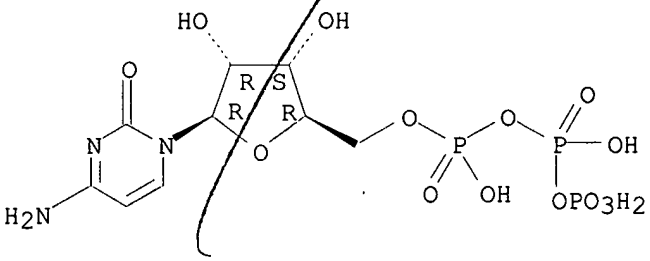
Searched by Barb O'Bryen, STIC 308-4291

AUTHOR(S): Swain, Judith L.; Sabina, Richard L.; McHale, Philip A.; Greenfield, Joseph C., Jr.; Holmes, Edward W.
CORPORATE SOURCE: Howard Hughes Med. Inst. Lab., Duke Univ., Durham, NC, 27710, USA
SOURCE: Am. J. Physiol. (1982), 242(5), H818-H826
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Brief coronary occlusions (12 min) were produced in 7 open-chest dogs, and repetitive myocardial samples were taken in order to det. the response of the nucleotide pool to ischemia and reperfusion. During ischemia, heart ATP levels decreased to 57% of control, and similar decreases occurred in the GTP, CTP, UTP, and NAD+ pools. The decrease in nucleotides was accompanied by an increase in nucleosides and bases. After 60 min of reperfusion, the content of all nucleotides had increased but was still less than nonischemic values. The content of nucleosides and bases decreased immediately upon reperfusion. In contrast, the creatine phosphate (CP) level fell to 10% of control during ischemia but rebounded to above control values immediately upon reperfusion. Apparently, the delayed nucleotide repletion is not caused by a defect in **mitochondrial** synthesis of ATP because CP content is restored rapidly. The slow repletion of nucleotides may be secondary to loss of nucleotide precursors during reperfusion and may result in widespread alterations in myocardial metab.

IT **65-47-4**
RL: BIOL (Biological study)
(of **heart**, in **heart ischemia** and reperfusion)
RN 65-47-4 HCAPLUS
CN Cytidine 5'-(tetrahydrogen triphosphate) (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 24 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1980:447066 HCAPLUS
DOCUMENT NUMBER: 93:47066
TITLE: Partially thiolated poly(cytidylic acid). Chemical modification of a preformed nucleic acid
AUTHOR(S): Bardos, Thomas J.; Novak, L.; Chakrabarti, P.; Ho, Y. K.
CORPORATE SOURCE: Sch. Pharm., State Univ. New York, Buffalo, NY, 14214, USA
SOURCE: Nucl. Acid Chem. (1978), Volume 2, 881-4. Editor(s): Townsend, Leroy B.; Tipson, R. Stuart. Wiley: New York, N. Y.
CODEN: 42TBAU
DOCUMENT TYPE: Conference
LANGUAGE: English

AB K poly(cytidylate) was converted to cetyltrimethylkammonium poly(cytidylate), which was stirred with MeOBr in MeOH 30 min at 0.degree., AcNMe2 and **NaSH** were added and the soln. was stirred for 1-1.5 h to give partially thiolated poly(cytidylic acid), which was

isolated as the Na salt. The product contained 8-12% 5-mercaptcytidylate units.

IT **68316-63-2P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and thiolation of, with sodium hydrosulfide)
RN 68316-63-2 HCAPLUS
CN 5'-Cytidylic acid, ion(1-), homopolymer, N,N,N-trimethyl-1-hexadecanaminium (9CI) (CA INDEX NAME)

CM 1

CRN 6899-10-1
CMF C19 H42 N

$Me_3^+N-(CH_2)_{15}-Me$

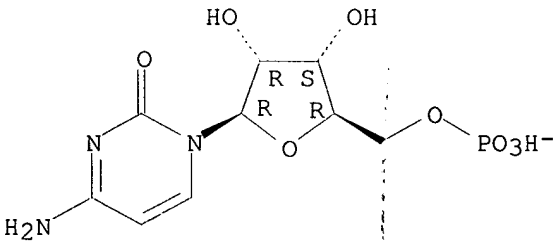
CM 2

CRN 68316-62-1
CMF (C9 H13 N3 O8 P)x
CCI PMS

CM 3

CRN 47151-23-5
CMF C9 H13 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.

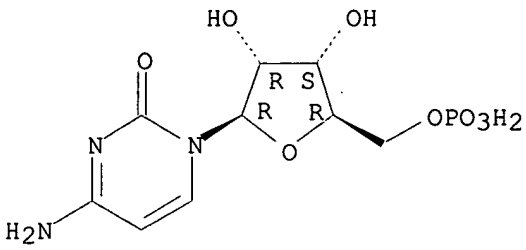


IT **30811-80-4DP**, partially thiolated
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of)
RN 30811-80-4 HCAPLUS
CN 5'-Cytidylic acid, homopolymer (9CI) (CA INDEX NAME)

CM 1

CRN 63-37-6
CMF C9 H14 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.



IT **26936-40-3**
RL: RCT (Reactant)
(reaction of, with cetyltrimethylammonium bromide)
RN 26936-40-3 HCAPLUS
CN 5'-Cytidylic acid, homopolymer, potassium salt (9CI) (CA INDEX NAME)

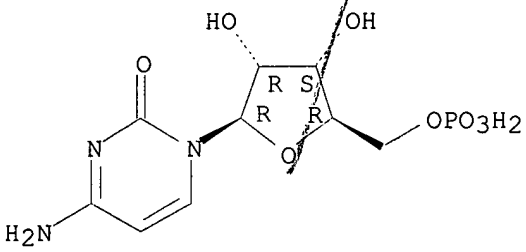
CM 1

CRN 30811-80-4
CMF (C9 H14 N3 O8 P)x
CCI PMS

CM 2

CRN 63-37-6
CMF C9 H14 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.



L107 ANSWER 25 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1979:417914 HCAPLUS
DOCUMENT NUMBER: 91:17914
TITLE: Vasogenic **cerebral edema**. Changes in membrane ATPases and correction by a phospholipid precursor
AUTHOR(S): Cohadon, F.; Rigoulet, M.; Guerin, B.; Vandendriessche, M.
CORPORATE SOURCE: Lab. Neuro-Chir. Exp., Univ. Bordeaux II, Bordeaux, F 33000, Fr.
SOURCE: Nouv. Presse Med. (1979), 8(19), 1589-91
CODEN: NPMDAD; ISSN: 0301-1518
DOCUMENT TYPE: Journal
LANGUAGE: French
AB In rabbits with cryogenic brain edema (induced with freezing of the right brain hemisphere in 4 points with liq. N2), the brain activities of mitochondrial membrane ATPase and Na,K-dependent ATPase of cell membranes decreased below initial values 1-4 days after the injury. In the exptl. rabbits, injection of 20 mg CDP-choline/kg daily beginning 24h after the

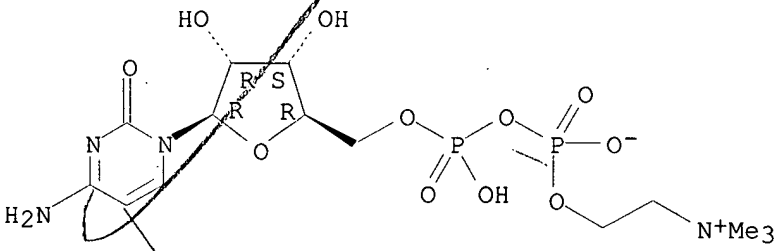
injury, improved or normalized the ATPase activities and reduced the edema.

IT **987-78-0**
RL: BIOL (Biological study)
(ATPase of mitochondrial membranes of brain in response to, in brain edema)

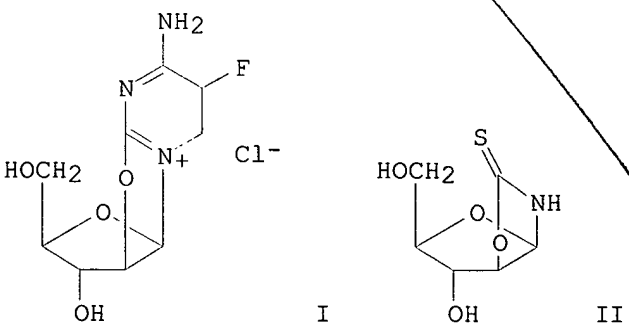
RN 987-78-0 HCAPLUS

CN Cytidine 5'-(trihydrogen diphosphate), P'-[2-(trimethylammonio)ethyl] ester, inner salt (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 26 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1979:6641 HCAPLUS
DOCUMENT NUMBER: 90:6641
TITLE: Fluorinated pyrimidine nucleosides. 2. Reaction of 2,2'-anhydro-1-.beta.-D-arabinofuranosyl-5-fluorocytosine hydrochloride with nitrogen and sulfur nucleophiles
AUTHOR(S): Cook, Alan F.; Holman, Michael J.
CORPORATE SOURCE: Chem. Res. Dep., Hoffmann-La Roche Inc., Nutley, N. J., USA
SOURCE: J. Org. Chem. (1978), 43(21), 4200-6
CODEN: JOCEAH; ISSN: 0022-3263
DOCUMENT TYPE: Journal
LANGUAGE: English
GI

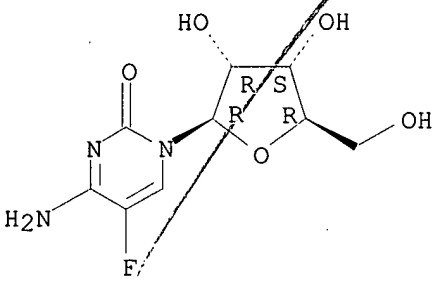


AB Reaction of the title nucleoside (I) with NH3 gave 1-.beta.-D-arabinofuranosyl-2,4-diamino-5-fluoropyrimidinium chloride by attack at C-2 of the pyrimidine ring. Reaction of I with MeNH2 gave the corresponding 2-methylamino deriv., which was rapidly converted into the 2,4-bis(methylamino)arabinoside by amine exchange at C-4. Treatment of I with EtNH2 or PrNH2 similarly produced the corresponding 2,4-bis(alkylamino) derivs. Reaction of I with MeNH2 for a prolonged reaction period resulted in rearrangement with loss of the sugar moiety to

produce 2-amino-5-fluoro-1-methyl-4-(methylimino)pyrimidine hydrohalide, the structure of which was confirmed by x-ray crystallog. Reaction of I with **NaSH** or H2S induced defluorination without cleavage of the anhydro bond to give 2,2'-anhydro-1-.beta.-D-arabinofuranosylcytosine; the oxazolidinethione II was also isolated as a byproduct. Treatment of the S and N-bridged analogs of I with **NaSH** also produced the corresponding defluorinated anhydro nucleosides.

IT **2341-22-2**
RL: RCT (Reactant)
(attempted defluorination of, with sodium hydrosulfite)
RN 2341-22-2 HCAPLUS
CN Cytidine, 5-fluoro- (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 27 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1978:615705 HCAPLUS
DOCUMENT NUMBER: 89:215705
TITLE: Partially thiolated poly(cytidylic acid). Chemical modification of a preformed nucleic acid
AUTHOR(S): Bardos, Thomas J.; Novak, L.; Chakrabarti, P.; Ho, Y. K.
CORPORATE SOURCE: Sch. Pharm., State Univ. New York, Buffalo, N. Y., USA
SOURCE: Nucleic Acid Chem. (1978), Volume 2, 881-4.
Editor(s): Townsend, Leroy B.; Tipson, R. Stuart.
Wiley: New York, N. Y.
CODEN: 39GCA6
DOCUMENT TYPE: Conference
LANGUAGE: English

AB Cetyltrimethylammonium poly(cytidylate) was treated with finely ground **NaSH**.2H2O to give the partially thiolated poly(cytidylic acid) after work up.

IT **68316-63-2P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and thiolation of)
RN 68316-63-2 HCAPLUS
CN 5'-Cytidylic acid, ion(1-), homopolymer, N,N,N-trimethyl-1-hexadecanaminium (9CI) (CA INDEX NAME)

CM 1

CRN 6899-10-1
CMF C19 H42 N

$Me_3^+N-(CH_2)_{15}-Me$

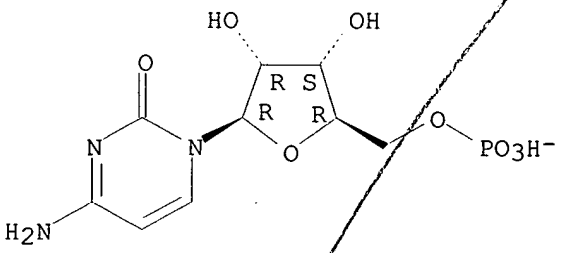
CM 2

CRN 68316-62-1
CMF (C9 H13 N3 O8 P)x
CCI PMS

CM 3

CRN 47151-23-5
CMF C9 H13 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.

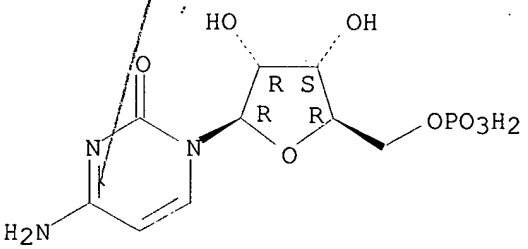


IT **30811-80-4DP**, thiolated deriv.
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of)
RN 30811-80-4 HCAPLUS
CN 5'-Cytidylic acid, homopolymer (9CI) (CA INDEX NAME)

CM 1

CRN 63-37-6
CMF C9 H14 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.



IT **26936-40-3**
RL: RCT (Reactant)
(reaction of, with cetyltrimethylammonium bromide)
RN 26936-40-3 HCAPLUS
CN 5'-Cytidylic acid, homopolymer, potassium salt (9CI) (CA INDEX NAME)

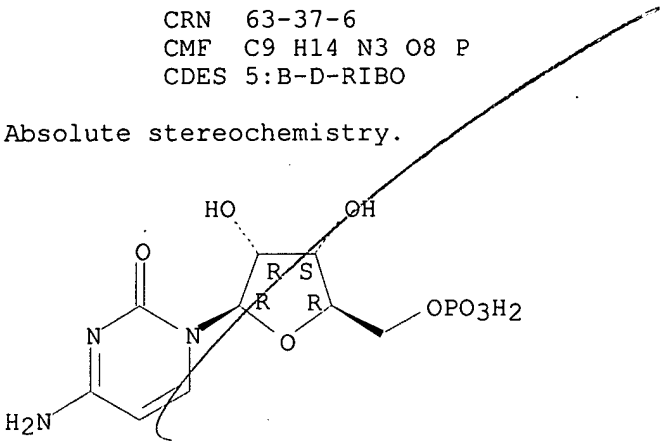
CM 1

CRN 30811-80-4
CMF (C9 H14 N3 O8 P)x
CCI PMS

CM 2

CRN 63-37-6
CMF C9 H14 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.



L107 ANSWER 28 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1978:580305 HCAPLUS
DOCUMENT NUMBER: 89:180305
TITLE: Sugar derivatives of purine compounds
INVENTOR(S): Isono, Kiyoshi; Azuma, Tsunemasa; Suzuki, Saburo
PATENT ASSIGNEE(S): Institute of Physical and Chemical Research, Japan
SOURCE: Japan. Kokai, 16 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

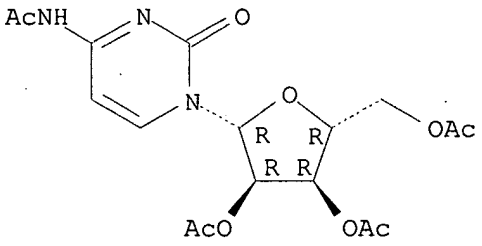
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 53056690	A2	19780523	JP 1976-117444	19760930
JP 55012917	B4	19800404		

AB Seven title sugar derivs., some of which are useful as remedies for **angina** pectoris and diseases caused by hormone imbalance (no data), were prepd. Thus, a mixt. of 206 mg tetraacetylcytidine, 288 mg N-benzoyl-N,9-bis(trimethylsilyl)adenine, and 0.1 mL SnCl4 in CH2Cl2-MeCN was refluxed 24 h to give 44.6% 2',3',5'-tri-O-acetyl-N6-benzoyladenosine, which was deacetylated to give adenosine.

IT **5040-18-6**
RL: RCT (Reactant)
(reaction of, with adenine deriv.)

RN 5040-18-6 HCAPLUS
CN Cytidine, N-acetyl-, 2',3',5'-triacetate (7CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



L107 ANSWER 29 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1977:400162 HCAPLUS

DOCUMENT NUMBER: 87:162
TITLE: Molecular approaches to inhibit oncogenesis by RNA tumor viruses
AUTHOR(S): Chandra, P.; Ebener, U.; Steel, Linda K.; Laube, H.; Gericke, D.; Mildner, B.; Bardos, T. J.; Ho, Y. K.; Goetz, A.
CORPORATE SOURCE: Abt. Molekularbiol., Gustav-Embden-Zent. Biol. Chem., Frankfurt, Ger.
SOURCE: Ann. N. Y. Acad. Sci. (1977), 284, 444-62
CODEN: ANYAA9
DOCUMENT TYPE: Journal
LANGUAGE: English

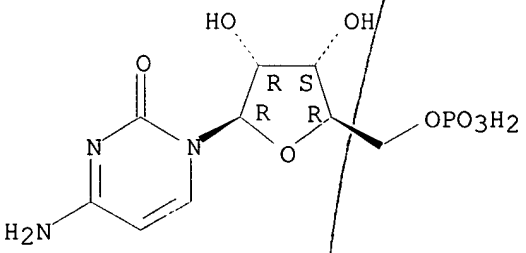
AB Mercaptanated polycytidylic acid (MPC), prepd. by treatment of polycytidylic acid with MeOBr, followed by reaction with **NaSH**, inhibited DNA polymerase [9012-90-2] from Friend leukemia virus, using either viral or synthetic nucleic acids as template. Similar inhibitory activity was given by partially thiolated tRNA and rRNA from Ehrlich ascites cells. MPC inhibited various viral, but not bacterial, DNA polymerases. MPC functioned as a dead template in the Friend leukemia virus DNA polymerase system, i.e., it interacted with the enzyme but failed to be transcribed. Prior incubation with MPC of cell-free spleen exts. from Friend leukemia virus-infected mice inhibited the splenomegaly obsd. in controls upon subsequent injection of the spleen ext. into other mice; further, cell-free spleen exts. prepd. from the splenomegaly-protected mice did not cause leukemia when injected into a 3rd group of mice, whereas mice inoculated with exts. from control mice developed leukemia.

IT **30811-80-4D**, mercaptanated
RL: BIOL (Biological study)
(DNA polymerase of Friend leukemia virus inhibition by)
RN 30811-80-4 HCAPLUS
CN 5'-Cytidylic acid, homopolymer (9CI) (CA INDEX NAME)

CM 1

CRN 63-37-6
CMF C9 H14 N3 O8 P
CDES 5:B-D-RIBO

Absolute stereochemistry.



L107 ANSWER 30 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1972:135834 HCAPLUS
DOCUMENT NUMBER: 76:135834
TITLE: Effects of adenosine and ATP on atrioventricular [AV] conduction and on AV junctional rhythm
AUTHOR(S): Urthaler, Ferdinand; James, Thomas N.
CORPORATE SOURCE: Sch. Med., Univ. Alabama, Birmingham, Ala., USA
SOURCE: J. Lab. Clin. Med. (1972), 79(1), 96-105
CODEN: JLCMAK
DOCUMENT TYPE: Journal

Searched by Barb O'Bryen, STIC 308-4291

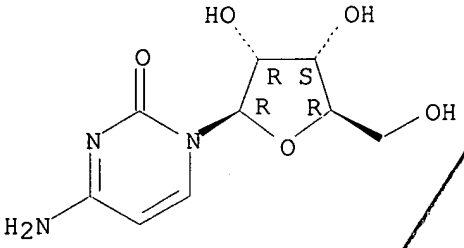
LANGUAGE: English

AB Of 18 ATP (I) [56-65-5] derivs. tested for electrophysiol. effects in open-chest anesthetized dogs by direct perfusion of the atrioventricular (AV) junctional region, all nucleotides except GTP [86-01-1] and cyclic GMP [7665-99-8] impaired AV conduction. I (1-10 mg/ml) produced an immediate **heart block** of 5-30 sec duration. Adenosine (II) [58-61-7] was less effective; no other nucleoside had any significant dromotropic activity. After selective suppression of the sinus node with eserine [57-47-6], I and II produced exclusively neg. chromotropic action at a concn. 1000-fold less than that required for the neg. dromotropic action.

IT **65-46-3 36051-68-0**
RL: BIOL (Biological study)
(heart atrioventricular conduction and rhythm in response to)

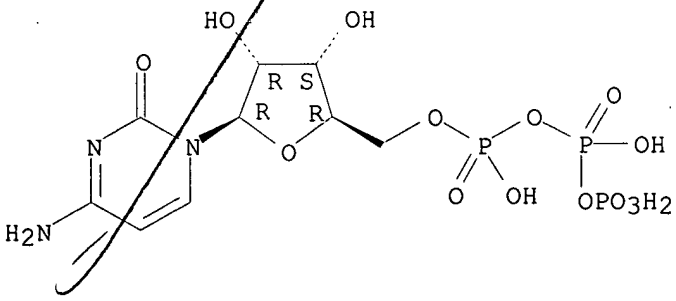
RN 65-46-3 HCAPLUS
CN Cytidine (8CI, 9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 36051-68-0 HCAPLUS
CN Cytidine 5'-(tetrahydrogen triphosphate), disodium salt (9CI) (CA INDEX NAME)

Absolute stereochemistry.



●2 Na

L107 ANSWER 31 OF 31 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1972:41808 HCAPLUS
DOCUMENT NUMBER: 76:41808
TITLE: Metabolism in **head injury**, with special reference to respiratory function to **mitochondria** of the **brain**, heart, liver, and kidney
AUTHOR(S): Izawa, Shiro
CORPORATE SOURCE: Sch. Med., Nihon Univ., Tokyo, Japan
SOURCE: Nichidai Igaku Zasshi (1971), 30(5), 421-44
CODEN: NICHAS

Searched by Barb O'Bryen, STIC 308-4291

DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Prednisolone (I) [50-24-8] or CDP-choline (II) [**987-78-0**] partly restored the impairment of respiratory function of the cerebral **mitochondria**, induced by head injury in rabbits, by inhibiting the formation of uncouplers or removing them from the **mitochondria**. The respiratory control rate, oxidative phosphorylation ratio, and O uptake of the brain, liver, and kidney **mitochondria** were all decreased following the injury. The respiratory function of the cerebral **mitochondria** was partly restored by I (19 mg/kg, i.v.) or II (10 mg/kg, i.v.) when given for 1 week from the day of injury.
IT **1477-47-0**
RL: BIOL (Biological study)
(**mitochondria** phosphorylation and respiration after **head injury** response to)
RN 1477-47-0 HCAPLUS

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